The Journal OF THE FLORIDA MEDICAL ASSOCIATION

DOES NOT CIRCULATE

Vol. XXXVIII APRIL, 1952 No. 10

UNIVERSITY OF MICHIGAN
APR 15 1952
MEDICAL LIBRARY

IN THIS ISSUE

Surgery, Common Bile Duct
Alpheus T. Kennedy

Pulmonary Embolism
David A. Newman

His Name Was Mudd
An Editorial

OFFICIAL PUBLICATION OF THE
FLORIDA MEDICAL ASSOCIATION

Across the placental barrier

Chloromycetin®

"It has been demonstrated, in pregnant women at term, that chloramphenicol passes from the maternal to the fetal blood stream in one hour following its ingestion, that it there attains a concentration equal to three-fourths of that in the maternal stream, and that the blood concentrations of mother and fetus are relatively the same after two and one-half hours."[1]

Therapeutic concentrations of well tolerated CHLOROMYCETIN (chloramphenicol, Parke-Davis) in the fetal blood stream are easily obtainable "by the simple oral administration of the drug to the mother."[2] Investigators have suggested, therefore, the empiric use of CHLOROMYCETIN in such virus infections as atypical pneumonia, in an attempt to avoid fetal damage.[3] Results with CHLOROMYCETIN in two patients with typhoid fever during pregnancy were reported recently as "quite satisfactory."[1]

Bibliography: (1) Stevenson, C. S.; Glazko, A. J.; Gillespie, E. C., and Maunder, J. B.: J.A.M.A. *146*:1190 (July 28) 1951. (2) Scott, W. C., and Warner, R. F.: J.A.M.A. *142*:1331 (April 29) 1950. (3) Ross, S., and others: J.A.M.A. *142*:1331 (April 29) 1950.

CHLOROMYCETIN is supplied in the following forms:
CHLOROMYCETIN Kapseals,® 250 mg., bottles of 16 and 100.
CHLOROMYCETIN Capsules, 100 mg., bottles of 25 and 100.
CHLOROMYCETIN Capsules, 50 mg., bottles of 25 and 100.
CHLOROMYCETIN Ophthalmic Ointment, 1%, ⅛-ounce collapsible tubes.
CHLOROMYCETIN Ophthalmic, 25 mg. dry powder for solution, individual vials with droppers.

MEDICAMENTA VERA

Parke, Davis & Company
DETROIT MICHIGAN

THE JOURNAL OF THE FLORIDA MEDICAL ASSOCIATION

OWNED AND PUBLISHED BY THE FLORIDA MEDICAL ASSOCIATION

Volume XXXVIII **APRIL, 1952** **No. 10**

CONTENTS

SCIENTIFIC ARTICLES

Experiences in Surgery of the Common Bile Duct, Alpheus T. Kennedy, M.D. 697

Electrocardiographic Changes in Pulmonary Embolism, David A. Newman, M.D. 701

Medical Ethics of Today, Raymond R. Killinger, M.D. 712

Routine Laboratory Determinations in "Organic" Versus "Functional" Disease: Study of Two Hundred and Eleven Unselected Office Patients in An Internist's Practice, Bernard D. Ross, M.D., and Gertrud Ernst, Ph.D. 715

ABSTRACTS OF MEDICAL ARTICLES

Drs. Frederick H. Bowen and James G. Lyerly 721

EDITORIALS AND COMMENTARIES

His Name Was Mudd 724

"Doctor, What Plans Have You Made?" 727

American Medical Education Foundation 727

Apathy in Voting 728

New Tests in the Etiologic Diagnosis of Diabetes Mellitus 729

Graduate Medical Education 730

Medical Licenses Granted 731

Procedure for Establishing and Maintaining Cancer Clinics Adopted by Florida Cancer Council, Tampa, Florida, January 18, 1952 733

GENERAL FEATURES

Officers and Committees 722

Your Blue Shield 734

State Board of Health 735

Births and Deaths 736

New Members 736

State News Items 737

Wanted — For Sale 740

Component Society Notes 741

Obituaries 744

Schedule of Meetings 753

List of County Medical Societies, Presidents and Secretaries 754

This Journal is not responsible for the opinions and statements of its contributors.

Published monthly at Jacksonville, Florida. Price $5.00 a year; single numbers, 50 cents. Address Journal of Florida Medical Association, P. O. Box 1018 (Fla. Theater Bldg.), Jacksonville 1, Fla. Telephone 6-1571. Accepted for mailing at special rate of postage provided for in Section 1103, Act of Congress of October 3, 1917; authorized October 16, 1918. Entered as second-class matter under Act of Congress of March 3, 1879, at the post office at Jacksonville, Florida, October 23, 1924.

THE JOURNAL OF THE FLORIDA MEDICAL ASSOCIATION
PUBLISHED MONTHLY

Volume XXXVIII | Jacksonville, Florida, April, 1952 | No. 10

Experiences in Surgery of the Common Bile Duct

ALPHEUS T. KENNEDY, M.D.
PENSACOLA

Surgery on the common bile duct is extremely interesting and presents a challenge to the surgeon to give his best performance in order to secure desired results.

An operation on the common bile duct above the duodenum was first described by Kummell[1] in 1890 and then by Thornton[2] in 1891. McBurney[3] performed a transduodenal operation first in 1892 for a stone at the ampulla of Vater. He did not open the common duct above the duodenum. By 1898 he reported 6 cases with 5 recoveries. Kocher[4] first performed a transduodenal operation in 1894, cutting through the duodenum and into the common duct and not the sphincter. The common duct and duodenum were sutured in side to side anastomosis.

Actually there are no signs or symptoms which enable the surgeon to determine, prior to operating, what is the exact cause of disease in the biliary system. Many laboratory procedures have been devised and are in general usage which are a help in determining whether or not jaundice is of an obstructive type. These tests are merely adjuncts and occasionally cloud the issue and mislead one who leans too heavily on them. It is also true that disease of a serious nature may exist in the common duct without giving rise to jaundice or colic.

What then are the reasons for exploring the common bile duct?

1. Jaundice or a history of intermittent attacks of jaundice. Here the history and laboratory procedures must be carefully evaluated, particularly when jaundice is present during the present illness.
2. Dilated or thickened common bile duct.
3. Palpation of a stone in the common bile duct, or inability to determine the cause of a suspicious mass. Lymph nodes are often found in the gastrohepatic ligament, and care must be exercised in determining whether a suspicious mass is a stone in the common duct or a lymph node alongside of the common duct.
4. Multiple small stones in the gallbladder.
5. Noncalculous gallbladder and history of biliary colic.

Exploration of the common bile duct is not advocated every time the gallbladder is removed, but it should be carefully exposed and the decision based on a consideration of the findings in conjunction with the history. This evaluation is essential to avoid performing an unnecessary exploration of the common duct and, what is more important, to avoid leaving behind a pathologic condition in the common duct that has not been adequately dealt with.

Analysis of Series

During the year July 1, 1949 to June 30, 1950, while I served as Senior Surgical Resident at the Duval Medical Center in Jacksonville, 60 patients were operated upon for disease of the extrahepatic biliary system. Cholecystectomy alone was performed on 40 patients; cholecystectomy and choledocholithotomy on 8 patients; and choledochotomy alone on 5 patients. Three patients had duodenocholedochotomy. Four patients had cholecystojejunostomy. An exploratory operation only was performed on 2 patients, who were found to have atresia of the extrahepatic biliary ducts.

The causes of obstruction as found at operation were:

Common duct stone	11
Fibrosis of ampulla of Vater	5
Carcinoma of head of pancreas	4
Congenital defects	2
Cholangitis	1
Stone lodged at ampulla of Vater	1

Read before the Florida Medical Association, Seventy-Seventh Annual Meeting, Hollywood, April 23, 1951.

Surgical Treatment

How then does one approach and deal with the various causes of obstruction? Stones are removed by scoops, stone forceps and irrigations of warm saline solution through a supraduodenal opening in the common duct. Probes and graduated dilators are then passed gently along the common duct and into the duodenum. As a final precaution, it is my practice to pass a catheter down the common duct towards the duodenum. This catheter is then irrigated with warm normal saline solution, and if none of the fluid is returned, it is assumed that the duct is patent to the duodenum.

Occasionally when it was thought that a probe had been passed into the duodenum, the injection of saline solution into the catheter gave immediate return, and further investigation revealed either more stones or a pathologic condition at the ampulla of Vater.

Stones, fibrosis, polyps, adenomas and carcinomas have been reported as causes of obstruction at the ampulla of Vater. Stones, polyps and adenomas are simply removed. The treatment of carcinoma of the ampulla of Vater requires radical surgery.

Fibrosis of the ampulla can be treated by introduction of graduated Bakes dilators, but this is occasionally followed by more scarring. This condition is best treated by a combined supraduodenal and transduodenal approach. A plastic operation is performed on the ampulla by cutting the sphincter and leaving a T tube in the common duct with one limb extending into the duodenum. The T tube serves as a splint to prevent a recurrence of the scarring and fibrosis and can be left in place for many months. Cattell[5] developed a T tube with one limb long for this purpose, but often an ordinary T tube is of sufficient length to be serviceable. The T tube is irrigated daily with distilled water and remains clamped off after the first seven to ten days postoperatively. A T tube is also left in place after removing stones, polyps or adenomas. Force should not be used in trying to pass probes or dilators. False openings may be made as a result. Damage to the pancreas or pancreatic ducts may occur.

The treatment of obstruction due to carcinoma of the head of the pancreas is first to relieve the obstruction. Numerous operations have been devised for radical removal of the pancreas and surrounding structures. In the 4 cases in this series distant metastases were already present, and thus the situation was hopeless. The five year salvage is practically nil even in the most favorable cases. In the cases reported treatment consisted of cholecystojejunostomy. This simple procedure allowed digestion and afforded the patients palliation for their remaining days. It is important to note that the status of the common duct should be determined before removing the gallbladder that might be of use in joining the biliary system to the gastrointestinal tract.

The patient with cholangitis was treated with exploration and T tube drainage of the common duct. He had an acute cholangitis, which is often a rapidly fatal disease. With the aid of chemotherapy and supportive measures, however, he survived.

The 2 patients with congenital defects had atresia of all of the external biliary apparatus including the gallbladder, cystic duct, common duct and hepatic ducts. These patients were infants, who received only exploratory laparotomy and eventually died.

Thus it is evident that the various types of obstruction which one may encounter are varied and demand individual attention to arrive at the proper solution in any given case. While it is true that opening the common duct adds to the length of any operation and mobilization of the duodenum with transduodenal exploration of the common duct is even more surgery, to leave behind disease in the common bile duct inadequately treated is a matter of serious portent. As stated earlier, I do not advocate opening the common bile duct in every patient operated upon for gallbladder disease. On the other hand, it is the responsibility of the operating surgeon to determine that the extrahepatic biliary ducts are patent and give evidence of no pathologic condition requiring surgical intervention.

In discussing the operative procedure, it is well to state at the beginning that every surgeon has his own technic and that this is to be desired. Certain surgical principles, however, should be followed. The type of incision employed is usually a subcostal incision and, if carried just beyond the midline on the left, it gives excellent exposure, particularly on short obese patients. The round ligament should be cut and carefully ligated. On tall thin patients a right paramedian or right rectus incision is utilized.

The common duct and its junction with the cystic duct should be carefully exposed by sharp and blunt dissection. The duct is palpated for

stones, and often if the operating surgeon will leave his position and go to the other side of the table (to the patient's left side), stones can be detected which are not otherwise felt. Before the common duct is opened, additional information can be obtained occasionally by aspiration of the bile with a 21 gauge needle and a 20 cc. syringe. The bile is then injected on a clean sponge. If the duct is normal in appearance, if the bile is clear, and if the patient's history is not indicative of jaundice, the common duct is not explored.

When the decision to open the common duct has been made, it is either grasped with Allis clamps or two sutures of fine material are placed in the duct and a longitudinal opening made between the sutures.

The duct is aspirated, and stones are removed by scoops or stone forceps, or often can be milked back up the common duct until they can be grasped with a stone forceps. A flexible probe is introduced and gently guided to enter the duodenum. As previously mentioned, force should not be used because of danger of making a false opening or injury to contiguous structures. Graduated dilators are then introduced, and if all goes well, a catheter of appropriate size is used to lavage the hepatic and common ducts and the catheter is passed into the duodenum. The surgeon must make every effort to be certain that the biliary system is open and draining well before considering the operation completed.

If obstruction is met with, then the right lateral edge of the duodenum is freely mobilized. The duodenum is opened longitudinally and exploration of the ampulla of Vater and the common duct completed. The duodenum is closed transversely with an inner layer of 00 or 000 chromic catgut and an outer layer of interrupted 000 silk. The common duct is closed about the T tube with interrupted stitches of 000 chromic atraumatic catgut.

The T tube is allowed to drain for several days and is then clamped off gradually. A cholangiogram is made before removal of every T tube. If the patient is being treated for disease at the ampulla of Vater, the T tube is left in place for several months and irrigated daily.

For the purpose of illustration, 2 cases are described.

Report of Cases

Case 1. — On Aug. 21, 1949, an 81 year old white man was admitted to the hospital with the chief complaint of abdominal pain. The patient had had a cholecystostomy six months previously at this hospital. At that time the gallbladder was drained, and several stones were removed. A cholangiogram made through the drainage tube showed blocking near the ampulla of Vater. The patient had been well thereafter except for an operative wound hernia until forty-eight hours prior to admission when there developed severe pain in the right upper quadrant of the abdomen. This was followed by nausea and vomiting. The pain was colicky in type and radiated around the costal margin to the back. In the four hours immediately before admission, the pain had been steady and had increased markedly in severity.

The physical examination revealed an 81 year old white man who was well nourished and well developed. The patient tossed from side to side and only after the administration of narcotics was it possible to examine him. The abdomen was rigid. There was a yellow tint to the skin.

At operation, the gallbladder was ruptured near the fundus, around the old fistulous drainage tract. There was stenosis of the ampulla of Vater, and the common duct and cystic duct were dilated. The common duct was opened, and with the use of Bakes dilators the ampulla was dilated until a size 18 Bakes dilator would pass with ease. A T tube was placed in the common duct with one limb going into the duodenum. The gallbladder was removed, and the hernia was repaired.

The postoperative course was somewhat stormy. The T tube was left in place four months. The patient is asymptomatic at the present time.

Case 2. — On Feb. 12, 1950 a 60 year old white woman was admitted with the chief complaint of pain in the abdomen. For two days she had had pain in the right upper quadrant, nausea, vomiting, itching of the skin, clay-colored stools and dark urine. She had had a cholecystectomy at the Duval Medical Center twenty-nine months prior to admission. The common duct was explored at that time. Four months after the operation the patient began to have intermittent episodes of pain in the right upper quadrant, nausea, vomiting, light-colored stools and dark urine. These occurred every three to six months and for the last six months had been occurring every two months.

The physical examination revealed a slightly obese white woman whose skin had a yellow color and showed excoriation and irritation from scratching. The temperature was 100 F. The pulse rate was 100, and the blood pressure was 120 cystolic and 70 diastolic.

There was a well healed subcostal scar in the right upper quadrant. The remainder of the physical examination gave essentially negative results except that the left great toe was missing, having been removed previously for treatment of diabetic gangrene.

A review of the old hospital chart showed that the patient had had a cholecystectomy in October of 1947. The common duct was blocked by a stone at the ampulla of Vater. The stone was milked backwards and removed through the opening in the common duct. Many small stones were removed also. Bakes dilators were passed easily into the duodenum after all the stones had been removed. A cholangiogram showed that the dye went into the duodenum.

At operation, there was a stenosis of the ampulla of Vater, and dilators could not be passed into the duodenum. It became necessary to open the duodenum and incise the ampulla of Vater. A T tube was then placed in the common duct with one limb of the T tube passing into the duodenum.

The postoperative course was stormy. The T tube was allowed to remain in place for three months. The patient has had no further difficulty.

Conclusion

The various types of obstruction to the common bile duct which one may encounter are varied and demand individual attention in order to arrive at the proper solution in any given patient.

When a surgeon operates upon a patient for biliary disease, it is his responsibility to determine that the extrahepatic biliary ducts are patent and give evidence of no disease requiring surgical intervention.

A pathologic condition at the ampulla of Vater is best handled by a combined transduodenal and supraduodenal approach.

A series of 60 surgical cases of disease of the extrahepatic biliary system is reported. Two cases illustrating surgery of the common bile duct are described.

References

1. Kummell, H.: Deutsche med. Wchnschr. 16:237, 1890; quoted in Mahorner, H.: Supraduodenal and Transduodenal Exploration of Common Bile Duct, Ann. Surg. 129:766-776 (June) 1949.
2. Thornton, J. K.: Observations on Additional Cases Illustrating Hepatic Surgery, Lancet 1:763-764 (April 4) 1891.
3. McBurney, C.: Removal of Biliary Calculi from the Common Duct by the Duodenal Route, Ann. Surg. 28:481; 525, discussion 517-519, 1898.
4. Kocher, T.: Text-Book of Operative Surgery, 3d English edition, authorized translation from the 5th German edition by H. J. Stiles and C. B. Paul, New York, The Macmillan Company, 1911.
5. Cattell, R. B.: New Type of T-Tube for Surgery of Biliary Tract, Lahey Clin. Bull. 4:197-204 (Jan.) 1946.

810 North Palafox Street.

Discussion

DR. EDWARD JELKS, Jacksonville: Dr. Kennedy, in his excellent paper which lays so much stress upon thoroughness and care in surgery of the biliary tract, introduces one point around which I should like to group my remarks, namely, that of seeing the biliary tree. If you do not see it, perhaps you will observe some of the unpleasant experiences that I have noted during thirty-six years of practicing surgery.

About twenty-four years ago I received a severe reprimand which has helped me ever since. We did a cholecystectomy upon a 48 year old woman. A few small stones were present. The operation was so simple it did not appear that any trouble could arise later. If some of you remember him, I am sure you realize the critical eye of the surgeon who watched me perform that operation. Dr. Knox Simpson was standing at the table as a helpful critic. After removal of the gallbladder, in a few days there developed jaundice, which cleared up immediately. Everything went along all right for about four months and then jaundice reappeared. Realizing that postoperative stricture probably was present, we sent the patient to the late Dr. Judd at the Mayo Clinic.

Not long afterward, it was my pleasure to talk with Dr. Judd about this patient, and he said, "That was a funny thing I found at operation; the common duct was intact, but it was constricted from the junction with the cystic duct to the duodenum. How did it look when you saw it at operation?" I replied, "It felt all right, Dr. Judd." He repeated, "How did it look?" That admonition by a distinguishd surgeon has not for a minute escaped my memory at any kind of operation for disease of the gallbladder.

This case is an example of the treacherous so-called "simple gallbladder," one that is easily accessible, which the surgeon thinks is so readily removable that no damage can be done to the patient. So, he merely palpates the biliary tree and does not expose it to view. Since being careful to visualize the biliary tree, we have made some interesting observations about it. There may be various locations and degrees of narrowing of the lumen. At the present time we are observing 2 or 3 patients to see what is going to happen to their biliary ducts. We know they have constrictions which at operation were not sufficiently developed to cause jaundice and bile was passing into the gastrointestinal tract.

Not long ago we operated upon a doctor who had jaundice. The gallbladder was perfectly normal except for a few small stones, but the common duct looked quite like some one sees in infants with multiple congenital constrictions. Bile came through partially, but the whole tree was involved. If we had not been careful to dissect out and visualize the biliary tree, in spite of some difficulty in doing so, we would have completed the operation without having correct information about the bile ducts, and when jaundice developed later, necessitating another operation, we would have criticized ourselves, thinking that at the previous operation we had injured the biliary tree.

It is just as important to be sure there is nothing wrong with the biliary tract as to find a lesion in it. With the ducts exposed, the liability of trauma to it is minimal. So, look and do not palpate only.

DR. WILLARD F. ANDE, West Palm Beach: Dr. Kennedy has given us a comprehensive discussion of the indications, the operative technic and the findings to be expected in surgery of the common duct. I was happy to hear, along with Dr. Jelks, his re-emphasis on the exposure of the common duct at the time of a cholecystectomy. Happily, I have witnessed no bad effects when this was not done, but as Dr. Jelks has emphasized, symptoms may recur, to the embarrassment of the surgeon and to the great discomfort of the patient.

Since many cases of this type occur in aged patients, the problem of a "prophylactic" cholecystectomy and perhaps choledochotomy in these patients arises. Many of you here have had patients, followed along by the family physician, who perhaps have disease of the extrahepatic biliary system and also actual known stones, cholelithiasis, but in addition they have, perhaps, some cardiac disability or a renal impairment with slight elevation of nonprotein nitrogen and perhaps a moderate to severe degree of sclerosis. The family physician carries them on from year to year and perhaps without any serious complications, but we have no way of knowing how many of these patients die as a result of their cardiac or renal disease or some other complication, a cerebral accident or what not. When they come to you with stones in the common duct, with acutely inflamed gangrenous gallbladders, or perforated gallbladders, they are dehydrated from vomiting and are suffering from some degree of malnutrition and anemia. They are in pretty foul shape, perhaps toxic from a generalized peritonitis. In these days of modern anesthesia we have antibiotics, improved surgical technic, intravenous fluids, and the like, to thank for the lowered mortality in these patients.

Had the family physician, however, submitted the patient for consultation or survey at a time when the patient was relatively well, when the operation could have been planned at the time when the patient was in the best condition, I believe that many cases of perforated gallbladder or stones in the common duct in aged patients could have been avoided. Today, with more advanced anesthesia, with developments in technic, with antibiotics, I think that many of these patients should have an operation before they get into a serious condition and provide a mortality rather than a successful operation. Thank you for the opportunity to discuss this paper.

DR. KENNEDY, concluding: I desire to thank the discussers and to express appreciation of their emphasis of the various points. Occasionally in the group of patients Dr. Ande spoke of, the patient has been told that he is too old, that he is not in condition to stand an operation, and then you are faced with an emergency in a patient whose mental attitude is, "I'm not going to pull through." You who are surgeons know especially well how much the mental state of the patient counts, how much the confidence of the patient in the doctor means, and how much the feeling of the patient that he is going to get well helps. This is true in any patient regardless of age.

Electrocardiographic Changes in Pulmonary Embolism

David A. Newman, M.D.
West Palm Beach

Pulmonary embolism may or may not give rise to specific changes in the electrocardiogram.[1,2] Actually, because the lesion is primarily pulmonary and not cardiac, there may be no significant typical electrocardiographic changes at all; or, only one or two of the changes may be present in a given case.[3] Unless the genesis of these changes is understood, they can be most confusing and are then too often passed off as the vague and ill-defined "myocardial damage."

In only about 10 to 20 per cent of cases of pulmonary embolism is there a characteristic electrocardiographic pattern. Electrocardiographic examination is therefore not a good method of making the diagnosis. This must still be clinical, and remains primarily dependent on the old "high index of suspicion." Clinically, the most important clue in the diagnosis is a sudden onset of tachycardia and shock, with or without pain in the chest, usually in a patient at rest in bed, and particularly so either in the postoperative period, or during treatment for congestive failure.[4] Another important suggestive clinical clue is the onset of sinus rhythm in a patient previously experiencing fibrillation, in association with a sudden onset of suspicious thoracic symptoms. The classic manifestations of acute pain in the chest, bloody sputum, pleural friction rub, consolidation and effusion, and the too late Moses' and Homans' signs may never appear.[4]

A not uncommon picture is one characterized by slight pain in the chest, mild cough and slight fever, without any definite physical signs other than, perhaps, a few basal or axillary crepitant rales. This has often passed as a pulmonary "virus infection." When it occurs in a patient in congestive failure, and particularly if it clears up rapidly, pulmonary embolism must be considered in the diagnosis.[9]

Read before the Florida Heart Association, Sixth Annual Meeting, Hollywood, April 22, 1951.

Because the electrocardiographic changes caused by pulmonary embolism are not always specific, and may not all be present in a given case, the configuration of the tracings will vary from patient to patient.[3] When present, however, and interpreted correctly in the light of their electrical genesis, these changes can serve to support the clinical diagnosis. This role is particularly important in the differential diagnosis between pulmonary embolism and myocardial infarction, especially that of the posterior wall. Recently, since the use of precordial V leads has become more general, the picture of anteroseptal infarction may also occasionally enter the differential diagnosis.

Many varied interpretations of the mechanism of pulmonary embolism have appeared in the literature.[2,5,6] The electrocardiographic criteria of pulmonary embolism were originally described by McGinn and White[2,5,6,7] in 1935 at a time when the precordial Wilson leads were not yet in common use. These authors emphasized the presence of an S in standard lead 1, a Q in lead 3, and a negative T in lead 3, associated with depression of the ST segment in leads 1 and 2 and elevation of the ST segment in lead 3.[8]

Since then, with the more general use of the V leads, many additional and varied patterns have been described.[2,6,9] Some authors have emphasized the changes characteristic of acute cor pulmonale;[1] others have emphasized the changes caused by clockwise rotation of the electrical axis of the heart with shift of the transitional zone to the left.[6] No doubt both are significant.

Following the case reports, which illustrate some of the more typical changes in pulmonary embolism, I will summarize the present views regarding the genesis of these changes in the electrocardiogram, as well as the criteria presently accepted as typical.

This report is the result of a study of 45 cases of pulmonary embolism in which treatment was given during the past six years at the St. Mary's

CASE 1

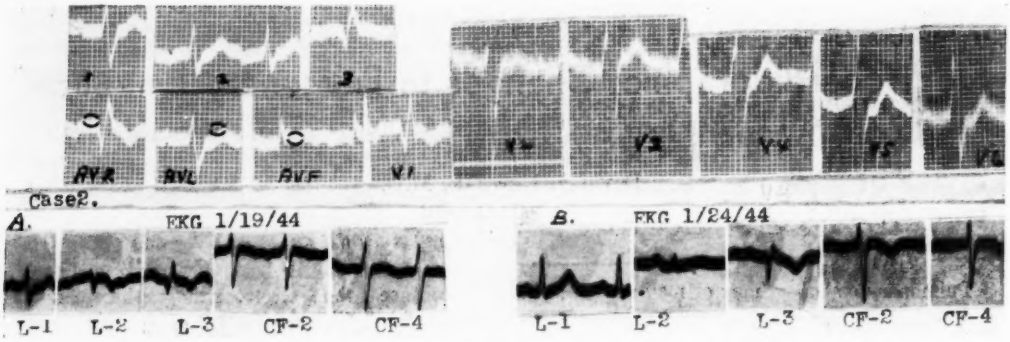


Fig. 1. — Case 1. Electrocardiogram taken twenty-three hours after the onset of pulmonary embolism. Deep S-1 and S-2, Q-3, elevated ST-3 and ST-AVF, late R in AVR and V-1, deep S in V-2 to V-6 and in VAL, small q and low diphasic T in AVF.

Case 2. (A) Electrocardiogram of Jan. 19, 1944, taken twenty-four hours after a third attack of pulmonary embolism. Broad S-1 and S-2, Q-3, negative T-3, depressed ST-1, elevated ST-3, deep S in CF-4, negative T in CF-2, diphasic T in CF-4.

(B) Electrocardiogram of January 24, on the same patient five days later. Pronounced diminution of S-1 and S-2, deeper negative T in CF-2 and CF-4.

and the Good Samaritan hospitals in West Palm Beach.

The purpose of the study was: (1) to correlate the electrocardiographic findings and diagnoses with the established diagnosis of pulmonary embolism, (2) to analyze the electrocardiographic changes present, in the light of the currently accepted criteria, and (3) to analyze the changes as to the mechanism that causes them.

Out of the 45 cases studied only those that presented unequivocal evidence in support of the diagnosis were selected for analysis. There were 21 such cases selected as the basis for this report. In all, one or more electrocardiograms were taken within twenty-four hours after the attack of pulmonary embolism. In 10 cases there were previous tracings for comparison.

The diagnosis in all these 21 cases was unequivocally supported by: (1) competent etiologic factors, (2) characteristic onset, symptoms, and signs, (3) characteristic course and progression, (4) adequate supportive laboratory and roentgen evidence, (5) electrocardiographic changes typical of pulmonary embolism in 8 cases and finally, (6) confirmation by autopsy in 5 cases.

Report of Cases

The 21 cases of substantiated pulmonary embolism were divided, on the basis of the electrocardiographic findings, into three groups:

I. Typical Group, Eight Cases

The electrocardiographic changes in this group were characteristic enough in pattern to make the diagnosis of pulmonary embolism possible. This is a larger percentage than the usually reported 10 to 20 per cent. Only 3 of these 8 cases (1, 4 and 5), however, were correctly interpreted at the time.

In summary, this group of 8 typical cases showed:

1. Evidences of clockwise rotation of the electrical axis of the heart, with shift of the transitional zone to the left, as seen in the precordial V leads. Depending on the position of the heart, this was transmitted to the limb and to the standard leads.
2. Evidences of right ventricular anoxia or ischemia causing T inversion and ST segment depression in the right precordial V leads.
3. Rapid reversion of these changes to normal.

Case 1.—J. A., a 67 year old man hospitalized on Oct. 28, 1949, had a positive blood Kahn reaction, hypertension, aortic insufficiency and cardiac enlargement. He was under treatment for congestive failure. There was a history of pain in the calf of the right leg on the night of admission. The onset on October 28 was typical, with acute pain in the chest, dyspnea, cyanosis, shock, syncope, tachycardia of 130, rales at the base of the right lung, and bloody sputum the next day; roentgen examination two days later showed "increased density of the lower half of the right lung field with probable pleural effusion." The patient improved at first, but died suddenly thirteen days after the onset in "acute respiratory failure."

The electrocardiogram of October 29 (fig. 1, case 1), taken twenty-three hours after the acute attack showed:

1. Deep broad S in leads 1 and 2.
2. Deep Q and negative T in lead 3.
3. ST segment elevation in lead 3 and in AVF.
4. QRS duration equal to 0.11—0.12 sec. (incomplete right bundle branch block).
5. Late R in V-1 and AVR.
6. Deep S in V-2 through V-6, and in AVL.
7. Small q and low diphasic T in AVF.

This case illustrates most of the features presently accepted as typical of pulmonary embolism.

It did not show the right ventricular "strain" pattern of T inversion in the right precordial leads, V-2 and V-3. It, however, did show the S-1, Q-3, negative T-3 pattern of McGinn and White and the pronounced clockwise rotation of the heart with shift of the transitional zone to the left.

The electrocardiographic diagnosis in this case was correctly reported as "probable pulmonary embolus."

Case 2.—L. D., a 72 year old woman admitted to the hospital on Jan. 19, 1944, had three episodes of pulmonary embolism in three weeks. Phlebitis of the right leg developed three weeks after an appendectomy for a gangrenous appendix. This was followed by three separate minor attacks of weakness with cold sweat and tachycardia. The third attack was also associated with pain in the chest, bloody sputum, fever, and a transient friction rub. Roentgen findings were confirmatory. The patient was discharged on February 2, and recovery was uneventful.

The electrocardiogram of January 19 (fig. 1, case 2A), twenty-four hours after the third attack, showed:

1. Tachycardia of 140.
2. Deep broad S in leads 1 and 2.
3. Deep Q-3 and negative T-3 (with late R-3).
4. ST depression in leads 1 and 2 (with so-called staircase ascent). ST elevation in lead 3 (with some coving).
5. Deep S in CF-4 suggesting clockwise rotation.
6. Negative T in CF-2 and low to diphasic T in CF-4, suggesting the right ventricular strain pattern.

The last two changes (5 and 6) are not as evident in the CF leads as they might have been in the V leads.

The electrocardiogram of January 24 (fig. 1, case 2B), five days later, showed:

1. Markedly diminished prominence of S-1 and S-2.
2. Deepening of the negative T in CF-2 and CF-4.
3. Q-3 and negative T-3 unchanged.

This case shows the typical McGinn-White pattern of S-1, Q-3, negative T-3 with ST depression in leads 1 and 2, and ST elevation in lead 3.

It also illustrates an important point in the differential diagnosis between pulmonary embolism and myocardial infarction of the posterior wall, which was missed here. The inversion of the T waves over the precordium, particularly over the right side of the heart (CF-2 and CF-4 in this case) does not occur in infarction of the posterior wall.[1] Rather, the T waves in the right precordial leads are reciprocally elevated and increased in amplitude.[8] They may remain unchanged, but are almost never inverted.[2] In pulmonary embolism they tend to become inverted, though this inversion may disappear in a short time.[10]

The electrocardiographic diagnosis in this case was "posterior wall infarction and (possibly) pulmonary embolism."

Case 3.—H. L. E., a 69 year old woman, had hypertensive arteriosclerotic heart disease and was in moderately severe congestive failure on maintenance doses of digoxin. She was admitted on Oct. 14, 1950 for pyelonephritis and renal calculus. Five days after admission she had a clinically typical attack of pulmonary embolism from which she recovered in seven days. Onset, course and all confirmatory findings were characteristic. She died three weeks later after nephrectomy on the left side.

The electrocardiogram of October 19 (fig. 2, case 3A), six hours after the attack, showed:

1. Prominent S in leads 1 and 2.
2. Deep Q and negative T in lead 3, and negative T in AVF.

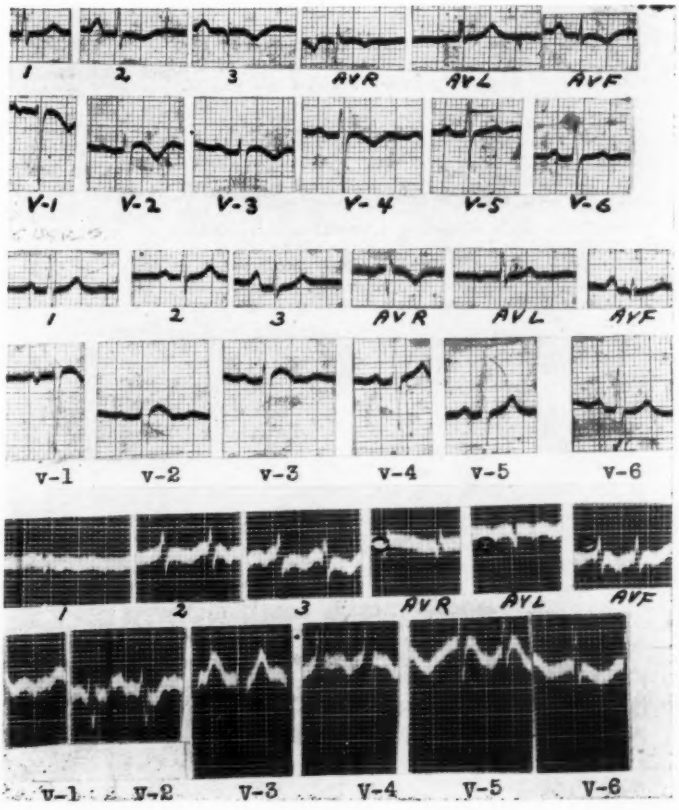


Fig. 2. — Case 3. (A)-10/19/50-Electrocardiogram taken on Oct. 19, 1950, about six hours after the onset of pulmonary embolism. S-1, S-2, Q-3, negative T-3, negative T in AVF and in V-1 through V-4, deep S through V-6, late r in AVR.

(B) - 10/25/50 - Electrocardiogram taken October 25 on the same patient. T-2 and T-3 up T-AVF up, T up in V-4, 5, 6, T less negative in V-2, 3, S smaller in V-6.

Case 4. — 3/6/50-Electrocardiogram taken nineteen hours after the onset of pulmonary embolism; auricular fibrillation. S-1, S-2, S-3, depressed ST-2 and ST-3, negative T in L-1, 2, 3, and AVF, negative T in V-4, 5, 6, late R and elevated ST in AVR.

3. Sharp T inversion in V-1 to V-4.
4. Deep S in V-1 through V-6 and in AVF.
5. Late R in AVR.

The electrocardiogram of October 25 (fig. 2, case 3B), six days later, showed almost complete reversion of some of the changes to normal. This was particularly true of the T waves in leads 2, 3 and AVF. The precordial T waves were now upright in V-4, 5, 6 and less negative in V-2, 3.

This case illustrates mainly (1) the typical right ventricular "strain" pattern, (2) the clockwise rotation of the electrical axis, as indicated by the shift of the transitional zone to the left, (3) the rapid reversion of the electrocardiographic changes to normal in contrast to those of myocardial infarction, and (4) the McGinn-White pattern in the standard leads.

The electrocardiographic interpretation in this case was "coronary heart disease, questionable apical infarct."

Case 4.—L. S., a 61 year old woman with pronounced hypertension of long standing, was under treatment for chronic congestive failure. Fibrillation had been present. At the onset of a clinically typical attack of pulmonary embolism there had been a transient change to regular sinus rhythm. She was admitted on March 6, 1950 and died three days later from mesenteric thrombosis. Autopsy showed two small recent pulmonary infarcts of the left lung, and a small recent apical myocardial infarct at the apex; also old rheumatic mitral and tricuspid stenosis.

The electrocardiogram of March 6 (fig. 2, case 4), nineteen hours after the attack, showed:

1. Auricular fibrillation.
2. Small s in lead 1, large S in leads 2 and 3, (but no Q-3).
3. ST slightly depressed in leads 2 and 3.
4. T negative in leads 1, 2, 3, and AVF.
5. T negative in V-1 and in V-4, 5, 6 (but not in V-2, 3).
6. Late R and elevated ST in AVR.

This case illustrates mainly the changes due to the clockwise rotation of the electrical axis, with shift of the transitional zone to the left, in a heart that was vertical despite long-standing pronounced hypertension.

Some of the changes due to the pulmonary infarction were partly obscured in this case by the coexistent apical myocardial infarct.

The electrocardiographic diagnosis was reported as "myocardial infarction, possible pulmonary embolus."

Case 5.—E. D., a 60 year old woman with recurrent thrombophlebitis, had the last attack two weeks before the pulmonary embolism, for which she was hospitalized on March 24, 1945. She had two separate attacks of pulmonary embolism clinically typical in onset and course, with recovery.

The electrocardiogram of March 26 (fig. 3, case 5A), six days after the first attack, showed:

1. Right bundle branch block with QRS of 0.12 sec.
2. Double-peaked widened R-R′ in CF-2 and CF-4.
3. Deep, broad large S in lead 1, small s in lead 2, (no Q-3), deep S in lead 3.
4. Late slurred widened R′ in lead 3.
5. T negative in leads 2 and 3 and sharply negative in leads CF-2 and CF-4.

The electrocardiograms of April 2 and 23 (fig. 3, case 5B and C), one and three weeks later, respectively, showed:

1. Complete disappearance of the right bundle branch block.
2. Development of deep S through CF-4 (due to clockwise rotation?)
3. The negative T waves in leads CF-2 and CF-4 persisted in this case.

This case illustrates the transient nature of the right bundle branch block often associated with pulmonary embolism.

The electrocardiographic diagnosis in this case was "probable pulmonary embolus."

Case 6.—B. C., a 71 year old man hospitalized on Nov. 9, 1950, was an arthritic with hypertensive cardiovascular disease and paroxysmal auricular fibrillation. The clinical onset of pulmonary embolism was atypical and silent. It was at first thought to be a "virus pneumonitis." The onset, however, was associated with a transient change to regular sinus rhythm. The subsequent course and studies, including the occurrence of embolic phenomena elsewhere, established the diagnosis.

The electrocardiogram of November 10 (fig. 3, case 6A), twenty-four hours after the acute episode, showed:

1. Auricular fibrillation.
2. Small s in lead 1.
3. Q and negative T in lead 3. (T negative in leads 1 and 2 as well.)
4. ST segment slightly depressed in leads 1 and 2 and slightly elevated in lead 3.
5. Deep S through V-5.
6. T inverted in V-4 through V-6 and AVL (ventricular "strain" pattern).

The electrocardiogram of November 13 (fig. 3, case 6B), three days later, showed:

1. Reversion to sinus bradycardia.
2. Disappearance of all the abnormalities mentioned.

This case illustrates (1) the association of pulmonary embolism with transient auricular fibrillation occurring often during the transition from auricular fibrillation to sinus rhythm, and (2) the rapid reversion of the electrocardiographic changes to normal.

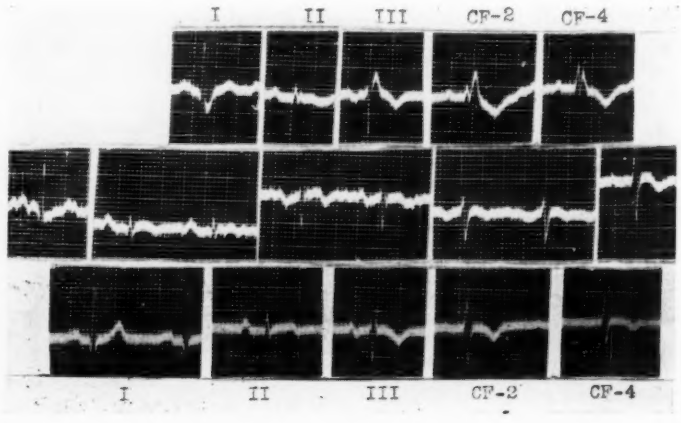


Fig 3.—Case 5. (A)-3/26/45-Right BBB with QRS of 0.12 sec., double peaking of R-R′ in CF-2 and CF-4, S-1, s-2, S-3, wide R′ in L-III, negative T in L-II, III, CF-2 and CF-4.

(B)-4/2/45-One week later, no bundle branch block, deep S through CF-4.

(C)-4/23/45-No change.

The electrocardiographic interpretation in this case was "myocardial damage, left ventricular strain."

Case 7.—K. P., a 69 year old woman, had a saphenous ligation on Nov. 14, 1950, followed by a nonhealing varicose ulcer and cellulitis. Eight days later she had a clinically typical onset of pulmonary embolism accompanied by dypsnea, tachycardia (with extrasystolic arrhythmia), shock, pains in the chest, bloody sputum the next day, and elevation of temperature to 101 F. Signs on physical examination and roentgen findings were confirmatory. The course was typical with rapid improvement and recovery, and recurrence of pulmonary embolism some time later, after discharge from the hospital on Jan. 6, 1951.

The electrocardiogram of November 22 (fig. 4, case 7A), four hours after the acute episode, showed:

1. Regular sinus rhythm, 92 per minute.
2. Prominent S in lead 1 (no S-2).
3. Deep and wide Q and negative T in lead 3 and in AVF.
4. ST segment slightly depressed in lead 2 and slightly raised in lead 3. Thus far the findings could be consistent with infarction of the posterior wall. In the V leads, however, there is:
5. Sharp late (low) inversion of T in the right precordial leads V-1 through V-4 (the right ventricular "strain" pattern). T is low upright in V-5 and V-6.
6. S is prominent through V-4, 5, 6 and AVL. There is a late small r in AVR. Both are indicative of clockwise rotation.

The electrocardiogram of November 24 (fig. 4, case 7B), two days after the attack, showed little change except, perhaps:

1. T in V-4 now upright.
2. S more prominent in V-4, 5, 6.

The electrocardiogram of December 15 (fig. 4, case 7C), twenty-three days after the attack, showed complete reversion of all changes to normal:

1. T waves in the right precordial leads V-2, 3, 4 are now upright.
2. S in leads 1, AVL, and V-5, 6 is now much less prominent.
3. Q and negative T in AVF are less pronounced. Those in lead 3 are the same as in an old tracing of eighteen months before.

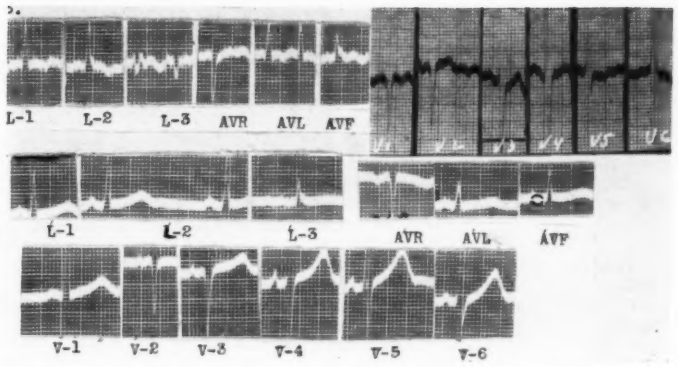


Fig. 3.—Case 6. (A)-11/10/50-Auricular fibrillations, s-1, Q-3, negative T-3, ST-1 and ST-2 down, ST-3 up, S through V-5, negative T in AVL and V-4 to V-6.

(B)-11/13/50-Sinus bradycardia, clearing of all abnormalities.

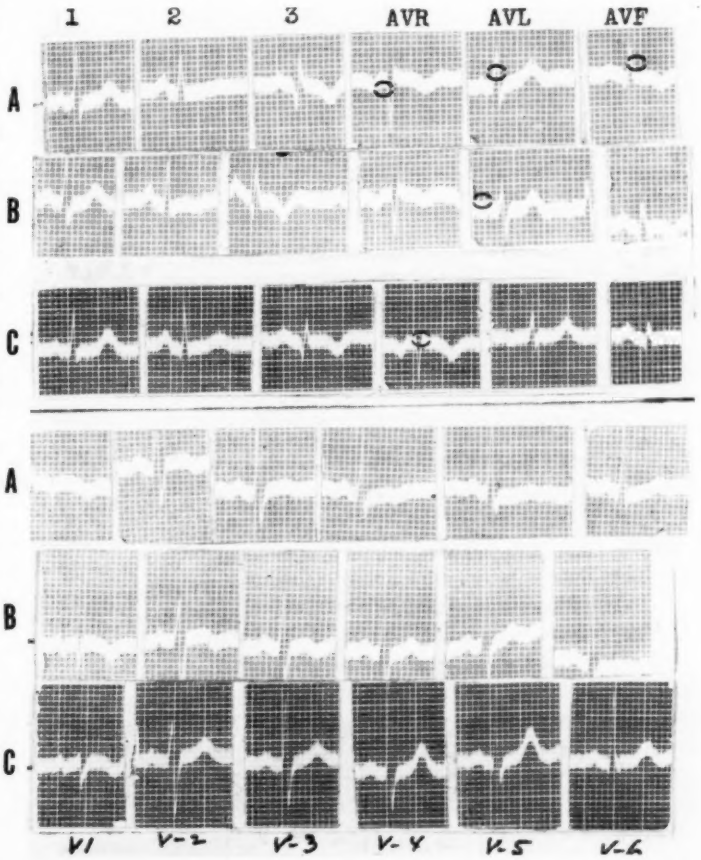


Fig. 4.—Case 7. (A) -11/22/50- S-1, Q-3, negative T-3, negative T-AVF, ST-2 down, ST-3 up, negative T in V-1 to V-4, S through V-6, S in AVL.

(B)-11/24/50-T up in V-4, S more prominent through V-6.

(C)-12/15/50-T up in V-2, 3, 4, s less prominent in L-1, AVL and V-5, 6, q in L-3 and AVF less pronounced.

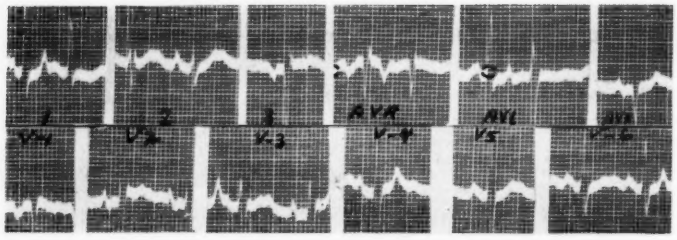


Fig. 5.—Case 8 -9/18/50- S-1, S-2 Q-3, diphasic T-3, Q-AVF, ST-1 and ST-2 down, ST-3 up, S through V-5, 6, late R in AVR.

This case again illustrates (1) the right ventricular "strain" pattern of inverted T waves in the right precordial leads, (2) their rapid reversion to normal in two days, (3) the McGinn-White pattern of S-1, Q-3, and negative T-3 in the standard leads, and (4) the pronounced clockwise rotation in the V leads.

At the time of its occurrence there was some question in the differential diagnosis between coronary thrombosis and pulmonary embolism. The electrocardiogram could have been materially helpful in making the differentiation.

The electrocardiographic interpretation, however, was "myocardial damage, posterior damage" and later "healing posterior infarct."

Case 8.—L. W., a 78 year old woman, had a typical attack of pulmonary embolism on Sept. 14, 1950, two days after an operation for cataract, with dyspnea, shock, cyanosis, and arrhythmic tachycardia of 150; the next day there was a temperature of 102 to 103 F., and a clinical diagnosis of consolidation (pneumonitis) at the bases of both lungs was made. Phlebitis of the veins of the leg was noted eight days later. There was rapid clearing of symptoms and signs in the chest. The patient died twelve days later from cerebral thrombosis. At autopsy, embolism of the right lower pulmonary artery with an infarct at the base of the right lung plus a smaller infarct at the base of the left lung was noted.

The electrocardiogram of September 18 (fig. 5, case 8), four days after the acute embolism, showed:

1. Large S in leads 1 and 2.
2. Deep Q and diphasic T in lead 3.
3. Deep Q in AVF.
4. ST depressed in leads 1 and 2 and slightly elevated in lead 3.
5. Deep S through V-5 and V-6 and late R in AVR indicating clockwise rotation, unexpected in a horizontal heart.

The expected T inversion in the right precordial leads was absent here and made the differential diagnosis more difficult.

This case illustrates mainly the clockwise rotation of the electrical axis of the heart that occurs in pulmonary embolism, unusual ordinarily in a horizontal heart.

The electrocardiographic interpretation in this case was reported as "myocardial damage."

II. Suggestive Group, Five Cases

The electrocardiographic changes in this group were not in a complete and typical pattern. At least three or more abnormalities suggesting pulmonary embolism, however, were present in each case. The diagnosis in only 1 case (12) in this group was correctly interpreted as "suspected pulmonary embolism." The electrocardiographic diagnoses in the remaining 4 cases were: case 9, "no myocardial damage;" case 10, "disease of the coronary arteries, low physiologic tone, low T waves in the V leads;" case 11, "serious myocardial damage, left ventricular strain, no occlusion;" and case 13, "large anterolateral infarct."

It is in this group that the correct electrocardiographic diagnosis is missed most often. Perhaps a better understanding of the mechanism involved would help to identify these changes more correctly, even when they are not in a complete and typical pattern.

Table 1 contains a summary of the abnormalities in this group of cases, as to their frequency of incidence.

Table 1. — Electrocardiographic Abnormalities in Five "Suggestive" Cases of Pulmonary Embolism

Abnormalities	Cases	Case Numbers
Deep S in lead 1	2	11,13
Deep S in lead 2	3	9,11,13
Q in lead 3	2	10,12
Negative T in lead 3	2	9,12
Negative T in lead 2	1	11
ST depression in lead 1	2	11,12
ST depression in lead 2	3	9,10,11
ST elevation in lead 3	2	9,12
Deep S in left V leads (clockwise rotation)	5	9,10,11 12,13
Transient BBB or impaired intraventricular conduction	2	9,13
Right "ventricular strain" pattern T inversion in V-1, 2, 3, (4) or CF2, CF4	3	10,11,13
Rapid reversion to normal (in cases with serial EKG's)	2	9,11
Q in AVF	3	10,11,12
Flat to negative T in AVF	3	10,11,12
Late R in AVR	1	13
ST elevation in AVR	1	11

Obviously, the table cannot indicate the sense of the combination of the particular abnormalities encountered in each case.[3] As can be seen from the table, however, the most frequent combination was the one indicative of clockwise rotation of the electrical axis of the heart with shift of the transitional zone to the left. The most significant was a combination of abnormalities that might at first be suggestive of infarction of the posterior wall, associated with the right "ventricular strain" pattern of T inversion over the right side of the heart in V-2, 3, (4).

Case 9.—A. C., a 71 year old man, was in good health except for hypertension. Acute onset of pulmonary embolism occurred on July 28, 1950, with drop in blood pressure, nausea, epigastric pain and cough. A small area of density on the roentgenogram over the dome of the

right diaphragm, suggestive of atelectasis secondary to infarction, was clearing three days later and was all clear in eleven days. The clinical course was confirmatory.

The electrocardiogram of July 29 (fig. 6, case 9), one day after the attack, showed:

1. Deep S through V-6, indicative of pronounced clockwise rotation in a heart that was electrically horizontal in position, in the face of known long-standing hypertension.
2. Prolonged intraventricular conduction time with a QRS of 0.10 to 0.12 sec.
3. Deep S' in lead 2, negative T in lead 3, with ST depression in lead 2 and ST elevation in lead 3.

Case 10.—T. N., a 48 year old woman, had bleeding uterine fibroids, recurrent attacks of thrombophelebitis and two previous attacks of pulmonary embolism elsewhere. Typical clinical onset of pulmonary embolism occurred at 2 a.m., on Jan. 1, 1949, with shock, tachycardia, dyspnea, pallor, pain in the chest and bloody sputum the next day. Signs and roentgen findings were confirmatory. Rapid improvement and clearing of signs followed.

The electrocardiogram of Jan. 1, (fig. 6, case 10), twelve hours after the acute attack, showed only the following:

1. Broad q and flat T in AVF.
2. Flat T in the right precordial V leads through V-4.
3. Prominent S through V-6.

The clockwise rotation unexpected in a horizontal heart, and the flat T waves in the right precordial leads indicative of right ventricular strain, might have been considered suggestive of pulmonary embolism.

Case 11.—C. E., a 64 year old man, was under treatment for congestive failure and was fully digitalized. Acute onset of pulmonary embolism occurred nineteen days after admission, on April 18, 1950, with shock, and hiccup. Roentgen and clinical findings were confirmatory. Rapid improvement and clearing of signs followed.

The electrocardiogram of April 18 (fig. 6, case 11), twelve hours after the acute attack, showed:

1. Deep S through V-6. This was absent in a tracing taken eight days previously, indicating development of clockwise rotation since then.
2. T inversion in V-2 to V-5.
3. S deep in standard leads 1 and 2.
4. ST depression in leads 1 and 2, probably due to digitalis.
5. Q and negative T in AVF.

The sudden development of clockwise rotation of the electrical axis in a heart that was in a horizontal position, and the right ventricular "strain" pattern in the presence of a Q and negative T in AVF, might have been considered suggestive of pulmonary embolism.

Case 12.—E. M., a 35 year old woman, had an attack of pulmonary embolism on July 1, 1950, seven days after a second stage thoracic sympathectomy for malignant hypertension with blood pressure of 200/130. The clinical onset was typical with violent cough, shock, cyanosis, dyspnea, tachypnea and tachycardia. Moist basal rales and increased P-2 were noted the next day. The course was characteristic, with rapid clearing and recovery. The patient went home fifteen days after the attack and did well.

The electrocardiogram of July 3 (fig. 7, case 12), two days after the acute attack, showed:

1. Q and negative T in lead 3 and in AVF.
2. Deep S through V-5 and S in V-6.
3. ST depressed in leads 1 and 2, in V-6 and in AVL, probably due to digitalis.

There is no T inversion here in the right precordial V leads, and no S in leads 1 and 2.

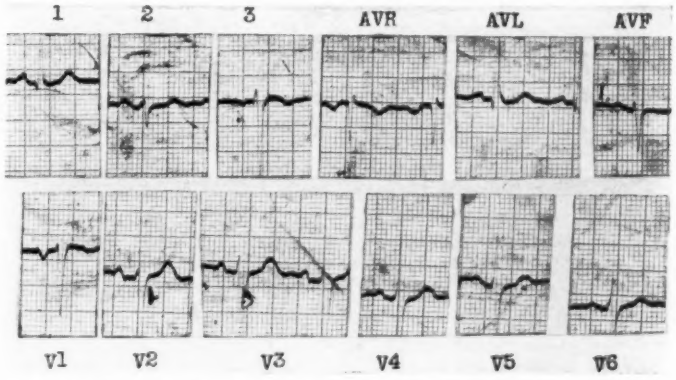


Fig. 6.—Case 9-7/29/50-Deep S through V-6, S-2, negative T-3, ST-2 down, ST-3 up, QRS 0.10 to 0.12 sec.

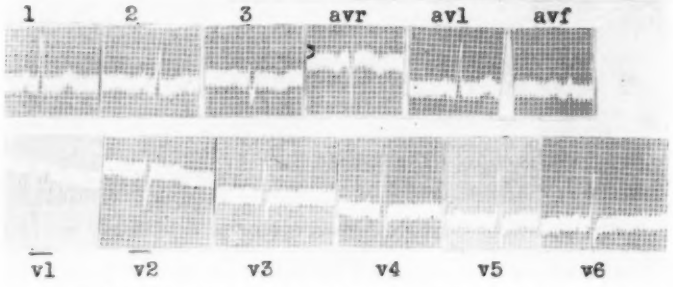


Fig. 6.—Case 10-1/1/49-Broad q and flat T in AVF, T flat in V-1-4, large S through V-6.

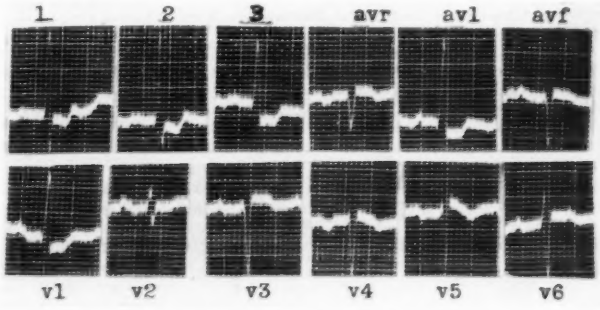


Fig. 6.—Case 11-4/18/50-Deep S through V-6, negative T in V-2 to V-5, S-1, S-2, ST-1 and ST-2 down, Q and negative T in AVF.

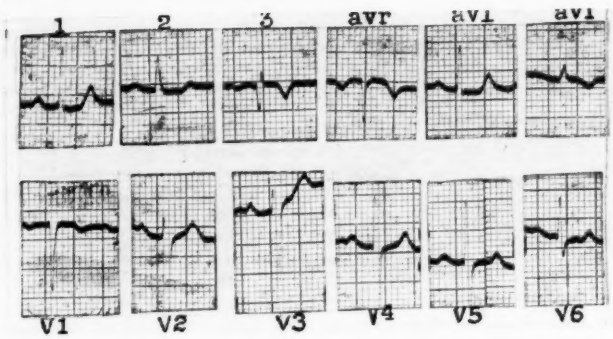
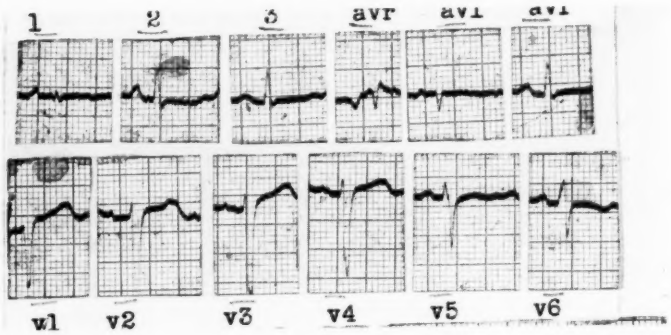
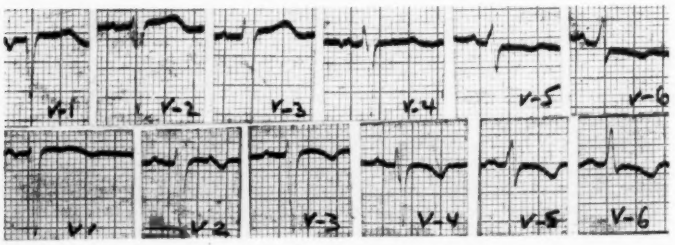


Fig. 7. — Case 12. Q-3, negative T-3, negative T in AVF, deep S through V-5, ST-1 and ST-2, and ST in AVL and ST in V-6 depressed.



Case 13. (A)—11/2/50—Small s-1, 2, 3, deep S in V-5, 6, T flat in 1, 2, 3, and AVL, negative in V-5, 6, widened QRS in V-2, 3.



(B)—11/4/50—S in V-6 less prominent, negative T in V-5, 6, more pronounced.

(C)—11/10/50—Negative T in all V leads.

The electrocardiographic interpretation in this case was correctly reported as "digitalis effect, myocardial damage, possible pulmonary embolus." The Q and negative T in leads 3 and AVF, associated with the tall upright T waves in the right-sided V leads might have warranted a diagnosis of infarction of the posterior wall. This case illustrates the difficulties in making a differential diagnosis between the two conditions.

Case 13.—W. F., a 53 year old man, had been in mild, untreated congestive failure, due to arteriosclerotic heart disease. There was a history of varicose ulcer of the leg, numerous sclerosing injections and unsuccessful venous ligation. Clinically typical onset of pulmonary embolism occurred on Nov. 2, 1950, preceded by cramps in the calf of the left leg earlier that day, with severe shock, cyanosis, transient loss of consciousness, tachycardia, and unobtainable blood pressure, followed next day by cough, bloody sputum and rales at the base of the right lung. The course was characteristic with rapid improvement, clearing of signs and recovery. The patient was discharged in nine days. The subsequent course was uneventful.

The electrocardiogram of November 2 (fig. 7, case 13A), twelve hours after the acute attack, showed:

1. Widened QRS in V-2 and V-3 with double peaking in some beats suggestive of right bundle branch block.
2. Deep S through V-5 and V-6 indicative of clockwise rotation.
3. Small s in leads 1, 2 and 3.
4. Flat T in leads 1, 2, 3 and AVL and inversion of T in V-5 and V-6.

No particular pattern could be elicited other than the suggestion of "myocardial damage."

The electrocardiogram of November 4 (fig. 7, case 13B), two days later (V leads only), showed:

1. S in V-6 less prominent.
2. T inversion in V-5 and 6 more pronounced.

The electrocardiogram of November 4 (fig. 7, case 13C), eight days later, showed:

1. T inversion in all the precordial leads, (as well as in the standard leads).

III. Atypical Group, Eight Cases

The electrocardiographic findings in this group showed no recognizable pattern in any way suggestive of a possible diagnosis of pulmonary embolism. In 5 of the 8 cases antecedent myocardial infarction served to distort the picture. In the remaining 3 cases the embolism was mild, transient and apparently not severe enough to cause any electrocardiographic changes at all.

Case 14.—T. McK., a 62 year old man admitted on Oct. 9, 1950, had had two known previous myocardial infarcts, one anterior and one posterior. While under treatment for congestive failure, he sustained a severe and clinically typical attack of pulmonary embolism associated with pleural effusion. Paracentesis yielded bloody fluid. He died one year later, and an old pulmonary infarct was found at autopsy.

The electrocardiogram of October 10, one day after the attack, failed to show any change that could be attributed to the pulmonary embolism. The antecedent tall T waves in the right-sided V leads, due to the previous posterior infarction, may have obscured the T inversion that the pulmonary embolism might have caused.

Case 15.—T. J., a 38 year old man, had a clinical onset and course not typical of pulmonary embolism; rather, it was one of left ventricular failure with acute pulmonary edema. Autopsy showed two old myocardial infarcts

(seven months old) as well as fresh infarcts of the upper lobe of the right lung.

An electrocardiogram, two days after the attack, showed the residuals of the previous anteroseptal infarction. The precordial T waves, however, which in an old tracing were upright, were now negative in V-1 to V-4.

Cases 16 and 17.—M. T., a woman aged 77, and F. B. M., a man aged 75, both had severe antecedent myocardial damage with multiple infarcts and pronounced residual T inversion throughout the electrocardiogram. The superimposed pulmonary embolism was obscured by these preexisting abnormalities. There were no typical or even suggestive changes of pulmonary embolism.

Case 18.—F. G. K., a 43 year old man, had an antecedent infarction of the posterior wall with residual Q and negative T in leads 2 and 3. In the precordials CF-2, CF-4 and CF-5 there were tall upright T waves. The superimposed fatal pulmonary embolism did not alter these changes to an extent sufficient to be identifiable.

Cases 19, 20 and 21.—In these 3 cases the condition was mild and transient. In 2, the pulmonary embolism occurred postoperatively, in 1 with transient fibrillation. In none of them did the electrocardiogram reveal any significant abnormalities.

I should like now to review briefly the mechanism causing the electrocardiographic changes in pulmonary embolism and the criteria presently accepted as typical.

Table 2. — Mechanism of Electrocardiographic Changes in Pulmonary Embolism

1. Myocardial Anoxia (Ischemia)
   - A. Reflex coronary vasoconstriction
   - B. Shock, drop in aortic blood pressure, diminished coronary blood flow
2. Acute Cor Pulmonale
   - A. Reflex pulmonary arteriolar vasoconstriction
   - B. Acute pulmonary hypertension
   - C. Acute increase in right intraventricular pressure
     - (1) Decrease in pressure gradient of blood flow to the right ventricle
     - (2) Dilatation and strain of the right ventricle and conus
     - (3) Increased work by the right ventricle
3. Changes in Electrical Position of the Heart
   - A. Clockwise rotation about long axis with shift of transitional zone to the left
   - B. Clockwise rotation about anteroposterior axis into more nearly vertical position.
4. Transient Right Bundle Branch Block

Mechanism[1,5,6,10,11]

The electrocardiographic changes of pulmonary embolism (table 2) are caused by:

1. Myocardial anoxia (ischemia) due to
   - A. Reflex coronary vasoconstriction resulting from the obstruction of a large branch of the pulmonary artery by an embolus. The existence of such vasovagal reflexes has been demonstrated experimentally.
   - B. Shock and drop in aortic blood pressure resulting in direct diminution of coronary blood flow.
2. Acute cor pulmonale resulting from
   - A. Reflex pulmonary arteriolar vasoconstriction in response to occlusion of a large branch of the pulmonary artery. It is conceivable that smaller emboli may fail to set up an intense enough reaction and therefore fail to cause significant electrocardiographic changes.
   - B. Acute pulmonary hypertension due to the vasoconstriction in the pulmonary circuit.
   - C. Sudden acute increase in right intraventricular pressure. This in turn causes:
     - (1) Decrease in the pressure gradient of blood flow to the right ventricle because 92 per cent of the venous return from it empties directly into the right ventricular chamber through the Thebesian veins.
     - (2) Dilatation and "strain" of the right ventricle and pulmonary conus with shift of the septum to the left.
     - (3) Increased work by the right ventricle in the face of a diminished blood supply.

     This process results in electrocardiographic changes confined mainly to the ST segment and T wave. It also causes:
3. Changes in the electrical position of the heart. The heart becomes rotated clockwise about both the anteroposterior and long axes, in such a way as to bring the right ventricle more anteriorly and to the left and place the heart in a more nearly vertical position. This rotation manifests itself in the electrocardiogram as a shift of the transitional zone to the left, with deep S waves as far as V-5 and V-6, in AVL, and in lead 1. It also causes a late R in AVR.
4. Transient right bundle branch block, a frequent result of the right ventricular ischemia, hypertension, dilatation and strain.

Not all of the components of this mechanism operate in every case. There are, therefore, variations from case to case.

Because reflex phenomena play a major role in this mechanism, the changes resulting from them are transient. This is one of the most notable characteristics of the electrocardiographic changes in pulmonary embolism — that they are transient and may revert to normal in a few days or even hours.

Table 3.—Diagnostic Criteria of Pulmonary Embolism

1. Standard Leads
   - Deep S-1, (or S-1 and S-2)
   - Prominent Q-3
   - ST-1 and ST-2 depression
   - ST-3 elevation
   - T-2 low, flat or diphasic
   - T-3 inverted
2. Precordial V Leads and AV Limb Leads
   - T inversion in V-1, 2, 3, (4)
   - Rapid reversion of negative T in V leads to normal
   - Deep S waves in V-4, 5, (6) and AVL
   - Transient right bundle branch block
   - Late r or r′ in AVR

The Diagnostic Criteria of Pulmonary Embolism (Table 3)[1,2,6,7,10,11]

I. In the standard leads

1. Deep S in L-1 (or L-1 and L-2)
   This S may be large and broad and suggestive of complete, or incomplete right bundle branch block.
2. Q present and prominent in L-3.
3. ST segment depressed in L-1 and L-2, with a steplike ascent.
4. ST segment elevated in L-3.
   The ST segment may often be short so that the T comes off directly from the QRS complex.
5. T is lowered, flattened or diphasic in L-2.
6. T is inverted in L-3.
   Items 1, 2 and 6 are the S-1Q-3 negative T-3 complex of McGinn and White.

II. In the precordial and AV limb leads

1. Consistent sharp inversion of the T waves in the V leads over the right precordial area — in V-1, 2, 3, (4).[2,11]
   This represents the so-called right "ventricular strain" pattern. Inverted T's may appear in all the V leads, but they appear first, and are most pronounced over the right side of the heart. They may not be reflected to the limb leads and therefore do not show up in the standard leads.
   In those cases in which there is not enough clockwise rotation of the heart to cause much change in the limb and standard leads, the T inversion in the right-sided V leads may be the only abnormal finding present.
2. Rapid reversion of the inverted T waves in the V leads to normal. This may occur within a short time, sometimes in only a few days. The inverted T waves in the V leads may become upright, usually first over the left ventricle and last over the right in V-1, 2, 3, (4).
3. Shift of the transitional zone to the left. This is due to the clockwise rotation causing the presence of deep S waves over the left precordium in V (4), 5, 6, and in AVL. This contributes to the formation of the prominent S in standard lead 1.
4. Transient right bundle branch block or delayed intraventricular conduction, with prolongation of the QRS interval and a late r′ or R′ in the right-sided V leads (V-1, 2).
5. Later R or R′ in AVR.
   This is also due to the clockwise rotation causing AVR to face the back of the heart. According to Goldberger,[10] it is due to the dilatation of the pulmonary conus as well. Right bundle branch block when present can also cause it. This late R or R′ contributes to the formation of the deep S in lead 1 (because L-1 is the reverse of AVR).
6. ST segment depression in the V leads and in AVL.
7. Elevation of the ST segment in AVR.
   This is due to myocardial anoxia as well as to the right "ventricular strain." It is often associated with:
8. Disappearance of the normally negative T in AVR.
9. Q and negative T in AVF are not characteristic findings, but occur often enough to cause confusion in the differential diagnosis from infarction of the posterior wall.

Differential Electrocardiographic Diagnosis Between Pulmonary Embolism and Posterior Wall Myocardial Infarction[2,6,11]

Both pulmonary embolism and posterior wall infarction can cause many of the changes listed, especially in the standard leads. Both can cause ST elevation in L-3, ST depression in L-1, in the V leads, and in AVL, Q-3, negative T-3, and Q and negative T in AVF. As a result, the changes may closely resemble each other and are often difficult to tell apart in a given single tracing; however,

there are some important differences (table 4):

1. S-1:
   It is most unusual to find a large S-1 in posterior infarction. In pulmonary embolism it is common.
2. Q-2, Q-3:
   In posterior infarction there is usually a Q in lead 2 as well as in lead 3. In pulmonary embolism there is usually no Q in lead 2.
3. ST-2 elevation versus depression.
   In posterior infarction ST-2 is usually elevated.
   In pulmonary embolism ST-2 is usually depressed.
4. Negative T-2, T-3.
   In posterior infarction T tends to be inverted both in lead 2 and lead 3. In pulmonary embolism T-2 tends to be lowered, flat, or diphasic, but is not characteristically inverted.
5. Rapid reversion versus progression.
   In pulmonary embolism the electrocardiographic changes are transient and revert to normal rapidly. In myocardial infarction the changes last longer and go through typical serial progression in successive tracings.
6. Sharp T inversion in V-1, 2, 3, (4) with rapid reversion to normal. This is the most important differential finding between the two. In pulmonary embolism it is the most important and consistent finding, representing the right "ventricular strain" pattern of cor pulmonale. In posterior infarction the exact opposite obtains. Here the T waves in the right-sided V leads are either normal or are reciprocally elevated and increased in amplitude in V-1, 2, 3, (4).

Anteroseptal infarction and digitalis can also cause T inversion in the right-sided V leads, V-1,-2, 3, (4). In the case of digitalis, the contour of the depressed ST segment here, as well as in the other leads, helps. In anteroseptal infarction the rotation of the heart is more likely to be counterclockwise rather than clockwise. The associated Q waves make the diagnosis.

Summary and Conclusions

A series of 21 cases of pulmonary embolism is reported in three groups, illustrating typical, suggestive and atypical electrocardiographic changes. The mechanism causing these changes, the diagnostic criteria and the differential electrocardiographic diagnosis between pulmonary embolism

Table 4.—Electrocardiographic Differences Between Pulmonary Embolism and Posterior Wall Infarction

	Pulmonary Embolism	Posterior Wall Infarction
Q-2	Q-2 may occur, but is uncommon	Q-2 is usual
Q-3	Present in both	
Q in AVF	May be present in both	
S-1	Deep and broad, is characteristic	Prominent S-1 not common
ST-1	Depressed in both	
ST-2	Depressed with steplike accent	Usually elevated
ST-3	Elevated in both	
ST in V leads and AVL	May be depressed in both	
T-2	Rarely inverted, may be low, flat or diphasic	Usually inverted
T-3	Inverted in both	
T in V-1,2, 3,(4)	Typically inverted	Typically up and reciprocally tall
Clockwise rotation (shift of transitional zone to left)	Typically present	Typically absent and is most unusual (unless anterior infarction is also present)
Late R in AVR	Typical	Rare
Progression	Rapid reversion to normal of some or all of these changes, particularly the rotation and T inversion in V-1,2,3,(4)	Typical progression of changes in serial tracings over period of weeks to months

and myocardial infarction of the posterior wall are reviewed.

Typical electrocardiographic changes of pulmonary embolism in complete combination occur in a minority of cases. No single abnormality is consistently present; however, in a considerable number of cases the findings are strongly suggestive of the mechanism as described, the most important of these being a combination of changes suggesting infarction of the posterior wall associated with the right ventricular strain pattern and pronounced clockwise rotation.

The severity and magnitude of the pulmonary infarction do not necessarily run parallel with the extent of electrocardiographic change and cannot be estimated from it. In milder cases, however, the smaller emboli may fail to cause any recognizable electrocardiographic changes.

When there are antecedent coronary disease and insufficiency, pulmonary embolism can cause enough myocardial anoxia to result in an electrocardiogram showing only disease of the coronary arteries. When there are antecedent myocardial infarcts, their electrocardiographic abnormalities obscure those of the superimposed pulmonary embolism and make it difficult or impossible to recognize them.

References

1. Wilson, F. N.; Rosenbaum, F. F., and Johnston, F. D.: Interpretation of Ventricular Complex of Electrocardiogram, Advances Int. Med. 2:1-63, 1947.
2. Wolff, L.: Electrocardiography: Fundamentals and Clinical Application, Philadelphia, W. B. Saunders Company, 1950, pp. 167-172.
3. Currens, J.: Electrocardiogram and Pulmonary Embolism, Proc. Staff Meet., Mayo Clin. 17:502-509 (Sept. 23) 1942.
4. Dressler, W.: Clinical Cardiology with Special Reference to Bedside Diagnosis, New York, Paul B. Hoeber, Inc., 1942, pp. 137-140.
5. Murnaghan, D.; McGinn, S., and White, P. D.: Pulmonary Embolism with and Without Acute Cor Pulmonale, with Especial Reference to Electrocardiogram, Am. Heart J. 25:573-597 (May) 1943.
6. Phillips, E., and Levine, H. D.: A Critical Evaluation of Extremity and Precordial Electrocardiography in Acute Cor Pulmonale, Am. Heart J. 39: 205-216 (Feb.) 1950.
7. McGinn, S., and White, P. D.: Acute Cor Pulmonale Resulting from Pulmonary Embolism; Its Clinical Recognition, J. A. M. A. 104:1473-1480 (April 27) 1935.
8. Carlotti, J.; Hardy, I. B., Jr.; Linton, R. R., and White, P. D.: Pulmonary Embolism in Medical Patients: Comparison of Incidence, Diagnosis and Effect of Treatment in 273 Cases at the Massachusetts General Hospital in Two Five-Year Periods (1936 to 1940 and 1941 to 1945 Inclusive), J. A. M. A. 134:1447-1452 (Aug. 23) 1947.
9. Durant, T. M.; Ginsburg, I. W., and Roesler, H.: Transient Bundle Branch Block and Other Electrocardiographic Changes in Pulmonary Embolism, Am. Heart J. 17:423-430 (March) 1939.
10. Goldberger, E.: Unipolar Lead Electrocardiography; Including Standard Leads, Augmented Unipolar Extremity Leads and Multiple Unipolar Precordial Leads, and a Section on Cardiac Arrhythmias, ed. 2, Philadelphia, Lea & Febiger, 1949, pp. 266-272.
11. Rosenbaum, F. F.: Clinical Electrocardiography, in Oxford Loose Leaf Medicine, Oxford University Press, pp. 370 (52-112) 1950.

Citizens Building.

Medical Ethics of Today

RAYMOND R. KILLINGER, M.D.
JACKSONVILLE

Doctors of Medicine, let us today rededicate ourselves to the proposition that in a great measure human health and happiness — yes, even existence — depend upon our thoughts and actions. This fundamental tenet of our profession has not changed since that greatest Healer of all time gave his life that others might live. The effectiveness of this proposition is compellingly reflected today through the magnitude of our innumerable devices in prolonging the span of life, and, as a corollary, increasing happiness. This inventory of correct principles of conduct between ourselves and the public has been going on since the first medicine man began studying the other fellow. Today I hope it culminates not so much in a conviction of sin or a recognition of danger as in an appreciation of the responsibility of our position. Personally, I find the responsibility tremendous, for I fully realize this gesture voices judgment of the most responsible group in the world.

In the beginning of this short talk I should like to reiterate the modernization of the Hippocratic Oath, known as the Declaration of Geneva 1948:

> Now being admitted to the profession of Medicine, I solemnly pledge to consecrate my life to the service of humanity. I will give respect and gratitude to my deserving teachers, I will practice medicine with conscience and dignity. The health and life of my patients will be my first consideration. I will hold in confidence all that my patient confides in me. I will maintain the honour and noble traditions of the medical profession. My colleagues will be as my brothers. I will not permit consideration of race, religion, nationality, party politics or social standing to intervene between my duty and my patient. I will maintain

Read before the Florida Chapter of the American Academy of General Practice, Second Annual Fall Meeting, Ocala, Oct. 14, 1951.

the utmost respect of human life from its conception. Even under threat I will not use my knowledge contrary to the laws of humanity. These promises I make freely and upon my honour.

In discussing "The Physicians' Responsibility" in the April 1951 number of the Journal of the Medical Association of Georgia, Dr. A. M. Phillips reminds us of a further obligation:

> We owe it to our patients to keep our skill at its peak by practicing, in our own expenditure of physical and mental energy, the moderation which we preach to them, and by utilizing the cultural and spiritual resources afforded by wholesome recreation, reading, home life, and worship. An extensive practice is exhausting; and when we become overtaxed our medical judgment wavers, our patients are denied their rightful consideration, and we become guilty of that assembly-line type of treatment which we so greatly deplore in England.

To render the greatest and most efficient service possible first requires thorough training on our part, and ever thereafter continued, competent training, as the study of medicine is for keeps. An explanation for increased specialization is the instinctive desire to do our utmost in striving for perfection. I have no uneasiness about the present controversy of specialist versus general practitioner. The fundamental truth is that both have a place in the sun, and, as usual, the laws of supply and demand will swing the pendulum to a proper proportion of each to fit the public needs. Each group is essential to the other, and the general practitioner no more desires condescension and flattery than the specialist deserves praise and adulation.

It has been said that the minds of men are held in bondage by the cardinal sin of certitude. Even with the rapid advancement of medicine today, there is yet present much of that drag on modern progress.

Teaching the medical teacher in the fundamentals of delivery and rhetoric is as much to be desired as competency in research, and, of necessity, the successful teacher must not only possess these attributes but be an excellent clinician as well. We must of course give the best in medical instruction, but we must also educate in the widest sense, for physicians now more than ever before must compare favorably with the members of other highly trained professions. In particular, I refer to a lack of interest in and aptitude for the preparation of scientific, political and community interest presentations, which are essential if we are to keep abreast of the broad aims of making Doctors of Medicine leaders in their communities.

Yes, we should be good doctors first and always. Today, however, we must have the knowhow to run efficiently a business office, knowledge of the Treasury Department's requirements as to deductible and nondeductible items, knowledge of social security and withholding taxes, knowledge of proper licensure, and knowledge that will serve as a broad guide to the new doctor in his selection of a locality and in getting established. Detailed instructions on these and many other phases of inter-relation medical ethics are fully outlined in "The Doctor" by Dr. Stanley R. Truman, published in 1951 by the Williams and Wilkins Company of Baltimore.

Organization

Naturally, one of the new physician's first acts is to align himself with the medical societies in his community and to go all the way through to the national levels. Only by organization can we obtain the necessary self improvement these bodies furnish. Thus, we learn to work with each other and for the public good, and to attain leadership and prestige in the public esteem.

The current united efforts of the medical profession have definitely slowed the Washington urge for the socialization of medicine. Contemplate your heroic work here in this state which was instrumental in expelling from Washington one of socialization's staunchest adherents. How well Benjamin Franklin's famous remark sums up our situation: "We must all hang together or we will hang separately."

In our zeal for organization, however, we are now encumbered by a myriad of meetings, conventions and conferences. These, I believe, should be streamlined and consolidated to conserve our time in the best interests of the public good.

Never must we neglect cordial and genuine relations with our strong allies, the dentist and the druggist. They are part and parcel of the same effort to heal the sick which engages us.

Our indispensable tool, the hospital, is now receiving much public disfavor because it is, in a measure, practicing medicine, and in effect running our business for us in its own way. This charge is serious and must be squarely met, for if physicians cannot practice medicine in the right way, their way, with their leadership, I pledge you the government will then take over the practice of medicine.

It would seem that now in all the world only one person is to be trusted — the Doctor. From the cradle to the grave, one calls continually upon him to testify. Not only does he attest one's advent into this world, but, once here, the individual

may not get married, get a job, change a job, leave the country, get insurance and meet a host of other can'ts and don'ts until he has the sanction of the doctor. Finally, he cannot even leave this mundane sphere until the doctor certifies for him that last passage west, and furthermore testifies as to what took him on that journey from which there is no return.

Insurance

Life is getting more complicated all the time. Its growing intricacies naturally suggest a brief discussion of the multitudinous insurance forms confronting us, which daily increase in volume and complexity. The best answer that I would have to this vexing problem is to hire a smart girl, for we can at least learn to sign our name on the right line. Seriously, however, this is a part of our life now. The man who is drawing compensation may have to feed a large family of children, and unless we promptly and cheerfully fill out his blanks, we give medicine a black eye. I think you get the hint about public relations with this pointed reminder.

Again, in the same vein, we doctors who have worked hard and rightfully for voluntary health insurance plans can be part of a conspiracy to wreck them. To do so is easy; only the doctor, the policy owner-patient and the hospital need connive, and the wreck will be quickly accomplished and fatal.

In all earnestness, I appeal to every member of the medical profession to be ever mindful that success or failure of voluntary health insurance lies in our hands. Do we want socialization of medicine? Then all we need do is to wreck the voluntary health insurance plans. To avoid such a catastrophe, how can we help the voluntary health plans to succeed? As general practitioners who handle the great bulk of insurance work, we especially owe it to our public that false statements as to present and past health are not concealed in the quest of something for nothing. Likewise, admissions and discharges, diagnostic, x-ray and laboratory work, as well as excessive hospital days, transfusions, medicines and the like, as a matter of convenience, are to be condemned.

The nub of the matter here is the necessity for which the reserves are set. Destroy needlessly this balance, and you jeopardize your future as a physician with freedom of choice in your hands. As physicians we have offered the public voluntary health plans as an alternative to compulsory health insurance. These plans cannot be as lucrative as investments in a similar field. If any company vitiates our intent honestly to protect its clients, then it is our plain duty, through the American Medical Association, to give our approval to the operation needs as we approve medical schools or drugs today.

Other Needs

The need for more and better nurses, as well as medical students, is apparent to all, as are more and better hospitals and allied appurtenances. None of these are to be obtained by wishful thinking or gullible promises from political aspirants with the proverbial axe to grind.

My friends and colleagues, we are going about this complicated problem the honest way. The medical profession through properly organized channels is making real sacrifices monetarily and by the sweat of the brow to promote and finance all of these needs, and more, that none should be lacking in proper medical care.

Truthfully, we can point to the irrefutable fact that we are the healthiest nation in the world. By our self-supporting attitude we are making the American public our staunch ally, as self reliance and individual thinking are distinctly American ways of life.

In conclusion, I could not do better in summarizing the main aspiration of medical ethics today than to quote from an address by Mr. Bernard M. Baruch:

> What is this adventure in health I see dawning, and towards which you all have been keeping the doctor's vigil through the night? This adventure, which you will have to lead — or it will fail — has many elements:
>
> 1. More and better doctors — in more places.
> 2. An immediate, complete survey to modernize medical education, with greater emphasis on chronic and degenerative diseases, mental hygiene, and preventive medicine.
> 3. More hospitals more evenly spread through the country.
> 4. Less specialists, more general practitioners.
> 5. Reorganize medical practice, stressing group medicine where needed and voluntary health insurance.
> 6. For those who cannot afford voluntary insurance, some form of insurance partly financed by the Government, covering people by law. I would call this "compulsory health insurance," if that term's proper meaning has not been lost.
> 7. Increased medical research.
> 8. Greatly expanded physical and mental rehabilitation.
> 9. Education to make health a national habit.
> 10. A vigorous, preventive medical program, reaching everyone, children above all.
> 11. A new cabinet post for health, education, social security.
> 12. Creation of a nonpolitical, watchdog committee to safeguard progress in medical care for veterans.

13. Increased numbers of well trained nurses and technicians.

14. Adequate dental care.

15. A stabilizing economy — inflation will make worthless any health program or anything else.

For your serious thought and consideration, I would leave with you these three questions:

1. Are the doctors going to do the practicing of medicine in the future?

2. Are the hospitals going to do the practicing of medicine in the future?

3. Is the government going to do the practicing of medicine in the future?

We of the medical profession now have the leadership. Let us make sure that we keep it and make the most of it. To do so — get in and pitch!

225 West Ashley Street.

Routine Laboratory Determinations in "Organic" Versus "Functional" Disease: Study of Two Hundred and Eleven Unselected Office Patients In An Internist's Practice

Bernard D. Ross, M.D.
and
Gertrud Ernst, Ph.D.
Miami

This paper presents objective evidence that physical abnormality can be found in a significant majority of medical patients, regardless of whether their symptoms appear "functional" or "organic" by usual clinical criteria. This is not consistent with current medical opinion, which estimates that one third to two thirds of all medical patients present primarily functional problems and thereby have no demonstrable physical basis for their complaints.[1]

Contained in this report are the results of a study of 211 unselected patients seen in the private practice of an internist in Miami between June 1, 1950 and June 1, 1951. All of these patients had at least one characteristic in common: they had symptoms which had caused them to consult a physician for relief. Patients simply seeking a "routine check-up" were not included. A parallel group of 21 "normal" controls was composed of volunteers who believed they were in good health and were certain that they did not require medical care. Excluded from this group of "normals" were any who did not pass a simple verbal screening test to minimize the likelihood of a need for medical aid and the possible existence of chronic subclinical sequelae to some acute disease suffered in the past (table 1).

From the Medical Research Unit, University of Miami, Miami, Fla.

Method

On the basis of history and physical examination, without consideration of laboratory findings, each patient was placed in one of three general categories: (1) Symptoms mainly due to "organic" disease. There were 65 subjects in this group, 24 males and 41 females. Ages ranged between the extremes of 13 and 80. Table 2 shows the age distribution, by decades, in this group as well as in the other groups. Table 3 is a list of the various diagnoses made in this group. The occurrence of multiple diagnostic entities in some patients caused the number of diagnoses to exceed the number of subjects in this group and in the following groups. (2) Symptoms due to "functional" problems superimposed upon obvious "organic" disease. Despite the presence of definite organic disease in this group of patients, their principal complaints were judged to have an appreciable "functional" component by usual clinical criteria. There were 53 subjects in this group, 22 males and 31 females. Ages ranged between the extremes of 15 and 75 years. Tables 4 and 5 are lists of the various "organic" and "functional" diagnoses made in this group of patients. (3) Symptoms primarily due

Table 1. — Criteria For Selection Of Normal Controls

No subject to be included who answers question 1 or 9 in the negative or any of the other seven questions in the affirmative.

1. Are you in good health as far as you know?
2. Have you had any illnesses, even a cold, during the past month?
3. Do you tire easily?
4. Are you very nervous?
5. Do you suffer from stomach trouble, constipation, gas, headaches, dizziness, palpitation or shortness of breath?
6. Have you ever had any serious illness, such as rheumatic fever, tuberculosis, malaria, nephritis, osteomyelitis, poliomyelitis, hypertension, jaundice, infectious mononucleosis or scarlet fever?
7. Have you suffered from allergic conditions such as asthma, hay fever or hives during the past year?
8. Have you had any operations or serious injury during the past year?
9. Do you sleep well?

Table 2. — Age Distribution by Decades

Group Number	Ages in Years	Male	Female	Total
1. "Organic"	10-19	1	3	4
	20-29	1	8	9
	30-39	3	4	7
	40-49	2	5	7
	50-59	9	9	18
	60-69	8	9	17
	70-79	1	1	2
	80-89	0	1	1
		25	40	65
2. "Organic" plus "functional"	10-19	0	1	1
	20-29	1	8	9
	30-39	5	4	9
	40-49	3	7	10
	50-59	9	8	17
	60-69	2	3	5
	70-79	1	1	2
	80-89	0	0	0
		21	32	53
3. "Functional"	10-19	0	1	1
	20-29	7	10	17
	30-39	14	22	36
	40-49	6	15	21
	50-59	2	10	12
	60-69	1	4	5
	70-79	0	1	1
	80-89	0	0	0
		30	63	93
4. "Normal" controls	10-19	0	1	1
	20-29	6	7	13
	30-39	2	0	2
	40-49	4	0	4
	50-59	0	0	0
	60-69	0	0	0
	70-79	1	0	1
	80-89	0	0	0
		13	8	21

Table 3. — Diagnoses in Patients With Symptoms Mainly Due to Organic Disease

Diagnosis	Cases
Arteriosclerotic heart disease	19
Chronic rheumatic heart disease	10
Hypertensive heart disease	8
Subacute virus infection	6
Chronic rheumatoid arthritis	4
Diabetes mellitus	3
Pregnancy	3
Chronic infectious mononucleosis	3
Hypertensive vascular disease	3
Chronic sciatic neuritis	2
Acute virus upper respiratory infection	2
Chronic viral hepatitis	2
Thyroid adenoma	2
Peptic ulcer	2
Chronic bursitis	1
Hyperthyroidism	1
Buerger's disease	1
Carcinoma of the bladder	1
Syphilitic heart disease	1
Hypothyroidism	1
Acute virus gastroenteritis	1
Brain tumor	1
Acute brachial neuritis	1
Subacute cholecystitis	1
Infectious polyarthritis (tuberculosis, arrested)	1
Amyotrophic lateral sclerosis	1
Cerebral arteriosclerosis	1
Chronic phlebitis	1
Cirrhosis of the liver	1
Disseminated lupus erythematosus	1
Multiple sclerosis	1
Latent tertiary syphilis	1
	87

Table 4. — Organic Diagnoses in Patients With Functional Symptoms Superimposed on Underlying Organic Disease

Diagnosis	Cases
Arteriosclerotic heart disease	18
Chronic rheumatic heart disease	7
Hypertensive heart disease	6
Chronic bronchitis	3
Peptic ulcer	3
Acute virus upper respiratory infection	2
Pregnancy	2
Pulmonary emphysema	2
Acute sciatic neuritis	2
Subacute virus infection	2
Chronic pelvic inflammatory disease	1
Chronic prostatitis	1
Hypothyroidism	1
Latent tertiary syphilis	1
Chronic intercostal neuritis	1
Spontaneous pneumothorax	1
Chronic rheumatoid arthritis	1
Subacute cystitis	1
Chronic infectious mononucleosis	1
Cirrhosis of the liver	1
Chorea	1
Chronic ulcerative colitis	1
Subacute bursitis	1
Hypertensive vascular disease	1
Parkinsonism	1
	62

Table 5. — Functional Diagnoses in Patients With Functional Symptoms Superimposed on Underlying Organic Disease

Diagnosis	Cases
Anxiety state, anxiety neurosis	28
Neurasthenia	19
Spastic colitis, irritable bowel syndrome	6
Reactive depression	3
Bronchial asthma	3
Hypochondriasis	2
Atopic dermatitis, neurodermatitis	1
Migraine headache	1
Chronic alcoholism	1
Tension headache	1
Neurocirculatory asthenia	1
	66

Table 6. — Diagnoses in Patients With Symptoms Primarily Due to Functional Problems

Diagnosis	Cases
Neurasthenia	37
Anxiety state, anxiety neurosis	23
Neurocirculatory asthenia	13
Reactive depression	11
Atopic dermatitis, neurodermatitis	11
Hypochondriasis	5
Vasomotor rhinitis	5
Spastic colitis	5
Migraine headache	3
Globus hystericus	3
Narcolepsy	1
Peptic ulcer syndrome	1
Tension headache	1
Paroxysmal tachycardia	1
Histamine headache	1
Mucous colitis	1
Pylorospasm	1
Bronchial asthma	1
Tic douloureux	1
	125

to "functional" problems. Patients in this group had no evidence of "organic" disease. Table 6 is a list of the diagnoses among these patients. There were 93 subjects in this group, 30 males and 63 females. Ages ranged between the extremes of 17 and 70 years.

There were 21 "normal" controls, 13 males and 8 females. Their ages ranged between 19 and 78 years.

Laboratory Examination. A urinalysis, complete blood count, erythrocyte sedimentation rate (Wintrobe, corrected), and cephalin flocculation test were performed on each subject. Each smear for differential count was carefully examined for the atypical lymphocytes and monocytes with vacuolated basophilic cytoplasm and fenestrated nuclei found in chronic infectious mononucleosis[2] and chronic viral hepatitis.[3] Two hundred leukocytes were counted each time such an examination was performed.

Individual laboratory determinations were considered abnormal in accord with the following criteria:

1. Sedimentation Rate: More than 12 mm. hr., after correction for hematocrit.
2. White Blood Count: Less than 5,000 or more than 10,000 per cubic mm.
3 Relative Lymphocyte Count: Percentage of lymphocytes above 38 per cent or below 20 per cent.
4. Atypical Lymphocytes and/or Monocytes: Presence of any such cells on examination of 200 leukocytes during course of differential count.
5. Cephalin Flocculation Test: At least 2 plus in twenty-four hours and at least 3 plus in forty-eight hours.

Results

A summary of the laboratory findings in the four groups of subjects is presented in figure 1.

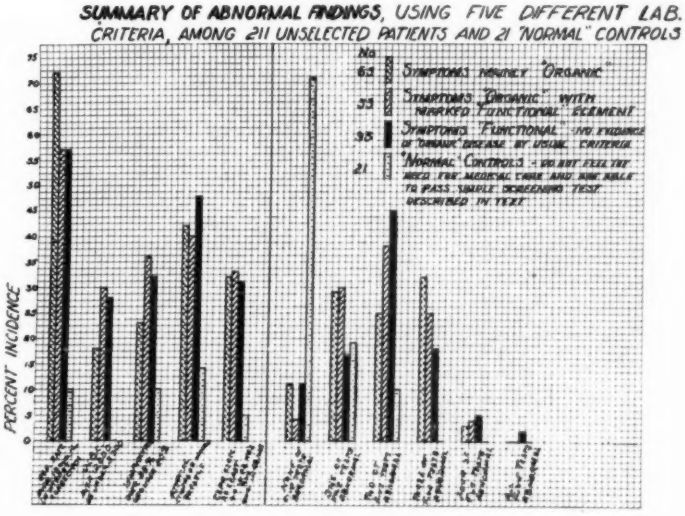


Figure 1

Sedimentation Rates. Sedimentation rates were elevated above 12 mm./hr. in 2 of the normal controls, an incidence of 9.6 per cent. A majority in each of the three groups of patients had abnormally elevated sedimentation rates. In group 1 ("organic" disease), the incidence of abnormal elevation was 72 per cent, in group 2 ("organic" disease with superimposed "functional" symptoms) 57 per cent and in group 3 ("functional" disease) 57 per cent. Figure 2 shows the relative distribution of sedimentation rates in each of the four groups.

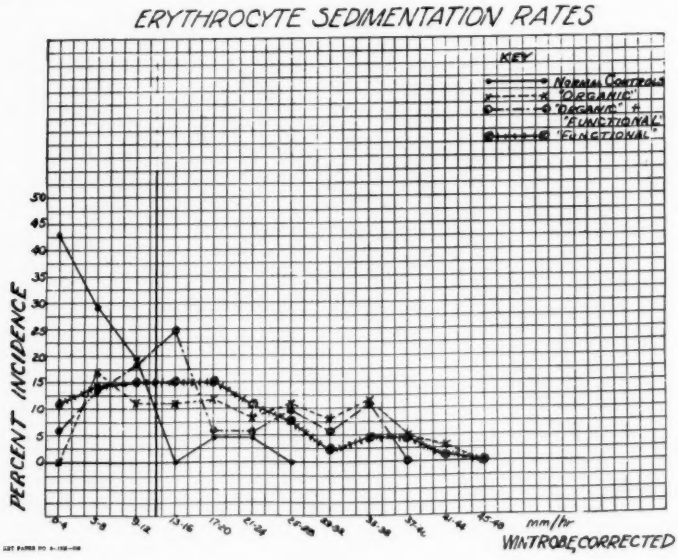


Figure 2

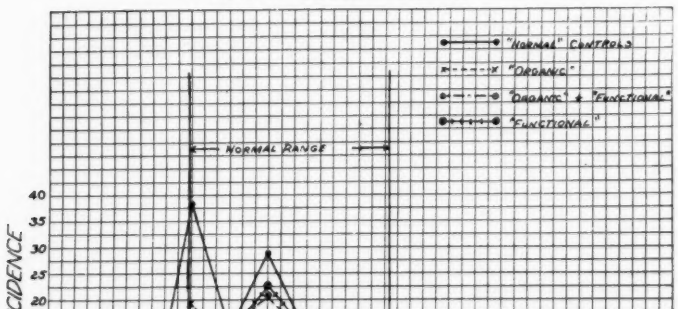


Figure 3

White Blood Counts. None of the normal controls had a count of less than 5,000 or more than 10,000. In patient groups 1, 2 and 3, the incidence of abnormally high plus abnormally low counts was 18 per cent, 30 per cent, and 28 per cent, respectively. Figure 3 shows the relative distribution of leukocytosis and leukopenia among the four groups of subjects.

Relative Lymphocyte Counts. Lymphocyte counts of more than 38 per cent were found in 2 of the normal controls, an incidence of 9.6 per cent. None of the normal controls had counts below 20 per cent. In patient groups 1, 2 and 3, the incidence of counts over 38 per cent or under 20 per cent was 23 per cent, 36 per cent and 32 per cent, respectively. Figure 4 shows the relative distribution of lymphocytosis in each of the four groups.

Atypical Lymphocytes and/or Monocytes. Atypical cells were found in 3 of the normal controls, an incidence of 14 per cent. The incidence of blood smears which showed these cells in the three groups of patients was 42 per cent, 40 per cent and 48 per cent, respectively. When atypical cells were found, they were present in concentrations varying from 0.5 to 10.0 per cent, which is comparable to the incidence of these cells reported by Isaacs[2] in chronic infectious mononucleosis. Figure 5 shows the relative percentage of atypical cells present in each of the four groups.

Cephalin Flocculation Tests. There was 1 positive reaction to the cephalin flocculation test among the 21 normal controls, an incidence of 4.8 per cent. In the three groups of patients, the incidence of positive reactions was 32 per cent, 33 per cent and 31

per cent, respectively. Figure 6 shows the relative distribution of degrees of positivity in this test in the four groups after twenty-four and forty-eight hours.

Distribution of Abnormal Findings Among Various Age Groups. It was thought necessary to study the possible significance of age differences as a factor influencing the validity of the results inasmuch as the "normal" controls included a larger proportion of subjects in the lower decades (especially the third) than did the three groups of patients. Table 7 shows the incidence of abnormal laboratory findings arranged according to decades. This presentation shows that the incidence of abnormal tests exhibits only minor variations among the various age groups, indicating that age difference is not a significant factor affecting the incidence of abnormal findings in these subjects. The incidence of abnormal reaction to tests in the 36 patients in the third decade was 1.9 per subject as compared to 0.5 in the 13 normal controls of comparable age. The over-all incidence of abnormal reaction to tests in the 211 patients was 1.9 as compared to 0.4 in the 21 normal controls. In the individual groups of patients, the incidence was 1.9 for group 1, 1.9 for group 2, and 2.0 for group 3.

Consistency in Degree of Variation of Abnormal Findings As Shown by Distribution Curves for Each of Five Different Laboratory Criteria. Comparison of figures 2, 3, 4, 5 and 6 reveals a consistent trend towards similarity among the distribution patterns in the patient groups as opposed to the appearance of those of the normal controls. This lends additional weight to the significance of the purely numerical incidence of abnormal values among the subjects, which is summarized in figure 1.

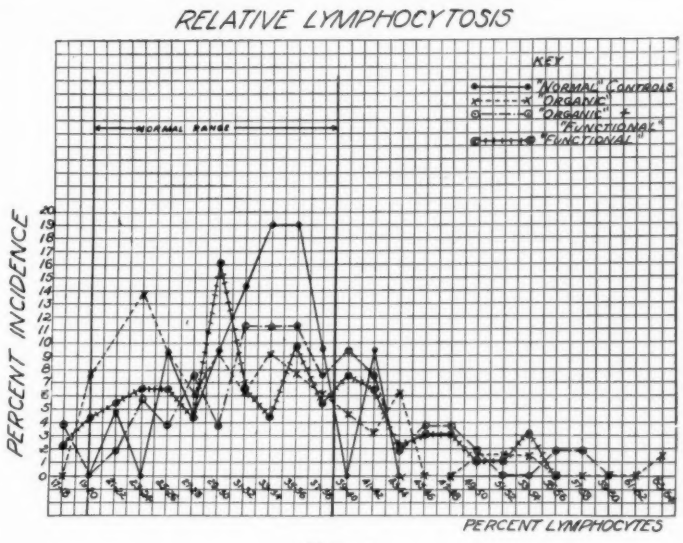


Figure 4

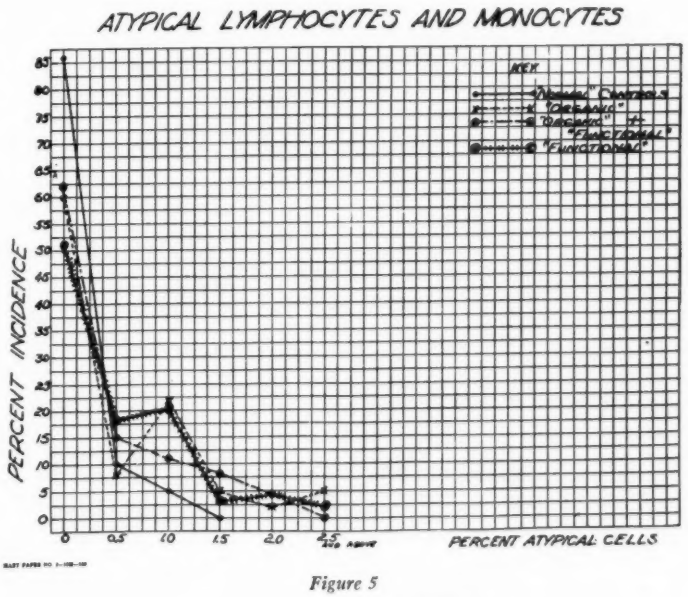


Figure 5

Comments

Our study of an unselected group of office patients indicates that the great majority had objective evidence of physical disease. Abnormalities in a simple series of five laboratory tests were present just as frequently in patients with a clinical diagnosis of primary "functional" disease as in those with "organic" disease or a combination of the two. In "normal" volunteers who did not feel the need for medical aid and had never had serious illnesses of the type which tend to have chronic sequelae, the incidence of abnormalities was much less.

Although we have been able to demonstrate the relatively constant association of objective abnormalities with "functional" symptoms in our series, we realize that we have no direct data to help us determine which is cause and which is effect. It may be that the changes we have described are primarily due to emotional wear and tear. But it has been shown by others that some of the objective findings we have described are observed in subjects who have chronic symptoms simulating psychoneurosis following an acute attack of viral hepatitis[3] or infectious mononucleosis.[2] Such symptoms respond better to therapy directed primarily towards the underlying physical disease rather than to psychiatric manifestations such as physical rest, dietary measures, removal of foci of infection and administration of adrenal cortical extracts.[2-4] Under such circumstances, abnormal laboratory findings disappear as symptoms subside, even though the latter may have initially seemed emotional in origin. It therefore seems more likely that the objective findings we have described are the cause rather than the result of the nervous instability occurring in the patients with "functional" symptoms.

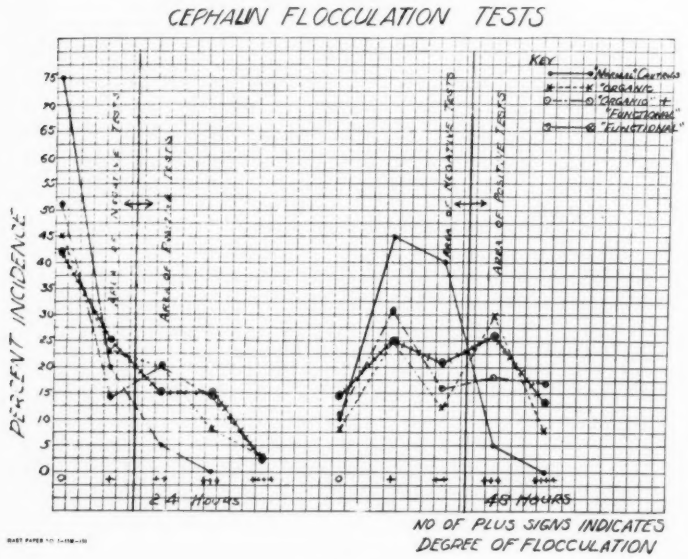


Figure 6

RELATIVE INCIDENCE OF ABNORMAL LABORATORY FINDINGS AMONG VARIOUS AGE GROUPS

Ages	Number of Subjects					Total Number of Positive Tests					Average Number of Positive Tests Per Subject				
	"Organic"	"Organic" plus "Functional"	"Functional"	Total Patients	"Normal" Controls	"Organic"	"Organic" plus "Functional"	"Functional"	Total Patients	"Normal" Controls	"Organic"	"Organic" plus "Functional"	"Functional"	Total Patients	"Normal" Controls
10-19	4	1	1	6	1	12	2	1	15	1	3.0	2.0	1.0	2.5	1.0
20-29	9	9	17	35	13	17	22	28	67	6	1.9	2.4	1.6	1.9	0.5
30-39	7	9	36	52	2	9	24	69	102	0	1.3	2.7	1.9	2.0	0.0
40-49	7	10	21	38	4	14	18	43	75	1	2.0	1.8	2.0	2.0	0.3
50-59	18	17	12	47	0	33	27	30	90	0	1.8	1.6	2.5	1.9	—
60-69	17	5	5	27	0	36	6	11	53	0	2.1	1.2	2.2	2.0	—
70-79	2	2	1	5	1	0	4	3	7	0	0.0	2.0	3.0	1.4	0.0
80-89	1	0	0	1	0	1	0	0	1	0	1.0	—	—	1.0	—
Total	65	53	93	211	21	122	101	185	410	8	1.9	1.9	2.0	1.9	0.4

Table 7

Consistent with this conclusion is the finding of great similarity in incidence and distribution of abnormal laboratory findings among the three groups of patients, as contrasted to healthy subjects.

Our findings strongly suggest that any distinction between "organic" and "functional" disease is artificial. They show that similar physiologic-pathologic disturbances can be detected in "organic" and "functional" disease by use of simple laboratory technics. These abnormalities may represent a common denominator which is the basis for the symptoms of instability occurring in patients with "functional" disease and may also be a prerequisite for the more obvious changes in tissue function and structure usually considered characteristic of "organic" disease.

Summary

Results are presented of the examination of 211 unselected patients in an internist's private practice as compared to 21 "normal" subjects. By routine use of the total white blood cell count, erythrocyte sedimentation rate, cephalin flocculation test and differential white blood cell count (with special care not to overlook atypical cells simulating

those seen in mononucleosis) it was shown that most patients had some objective evidence of abnormality, regardless of whether they had "organic" or "functional" disease by usual clinical criteria.

References

1. Wilbur, D. L.: Clinical Management of Patient With Fatigue and Nervousness, J. A. M. A. 141:1199-1204 (Dec. 24) 1949.
2. Isaacs, R.: Chronic Infectious Mononucleosis, Blood 3:858-861 (Aug.) 1948.
3. Barker, M. H.; Capps, R. B., and Allen, F. W.: Chronic Hepatitis in Mediterranean Theatre; New Clinical Syndrome, J. A. M. A. 129:653-659 (Nov. 3) 1945.
4. Koszalka, M. F.; Lindert, M. C. F.; Snodgrass, H. M., and Lerner, H. B.: Hepatitis and Its Sequelae, Including Development of Portal Cirrhosis; Observations on 100 Cases, Arch. Int. Med. 84:782-797 (Nov.) 1949.
5. Mote, J. R.: Proceedings of the First Clinical ACTH Conference, Philadelphia, The Blakiston Company, 1950.

1819 Biscayne Boulevard.

ABSTRACTS OF MEDICAL ARTICLES

The Determination of the Blood Amylase in Fluid Aspirated from the Flank in Pancreatitis. By Frederick H. Bowen, M.D. South. M. J. 44:775-777 (Sept.) 1951.

A case of chronic relapsing pancreatitis, with autopsy, is reported, in which the patient eventually died of acute hemorrhagic pancreatitis. In this case, a blood amylase determination on fluid aspirated from the area of bluish discoloration in the flank was obtained and proved to be a helpful diagnostic procedure. On this aspirated fluid the blood amylase determination was 4,210 units, and the control value was 177 units.

The author observes that the appearance of an area of bluish discoloration in the flanks in a person who has had severe abdominal pain for a day or two is a well known but infrequent sign of hemorrhagic pancreatitis and is almost always pathognomonic of this condition. The bloody extravasation tends to extend retroperitoneally and gravitate downward and laterally, becoming apparent as a greenish yellow or purple area in the loins lateral to the erector spinae muscles.

A review of the literature indicated that a blood amylase determination on fluid aspirated from the flank in acute pancreatitis has not previously been reported. In certain cases of pancreatitis, this new diagnostic aid should be of value.

Prefrontal Lobotomy for the Relief of Intractable Pain. By James G. Lyerly, M.D. Am. J. Surg. 81:526-532 (May) 1951.

A series of 12 cases of intractable pain relieved by bilateral prefrontal lobotomy is reported. Included are 3 cases of painful amputation stump, 3 cases of carcinoma of the pelvic region, 2 cases of atypical facial neuralgia and 1 each of severe intercostal neuralgia, pain of head, neck and arm following cervical disk removal, and post-traumatic headaches and tinnitus. In every case the bilateral prefrontal lobotomy was performed because the unilateral operation too frequently has been ineffective in relieving patients with mental disorders. The operation does not abolish physical pain, but tends to change the mental attitude so that the patient does not suffer as he did before. In like manner, the mood is elevated, and there is a lessening of nervous tension, anxiety and fear of the future. It is an easy matter to withdraw opiates even though drug addiction was a major factor in the illness.

Dr. Lyerly concludes that prefrontal lobotomy should not replace cordotomy and other surgical measures for the relief of pain in all cases. He adds that the patient and the family should help in making the decision as to the procedure of choice.

FLORIDA MEDICAL ASSOCIATION

OFFICERS AND COMMITTEES

OFFICERS

DAVID R. MURPHEY, JR., M.D., President.....*Tampa*
ROBERT B. MCIVER, M.D., Pres.-elect.....*Jacksonville*
FREDERICK K. HERPEL, M.D., 1st Vice Pres..*W. Palm Beach*
CHAS. J. COLLINS, M.D., 2nd Vice Pres.....*Orlando*
EDWARD R. ANNIS, M.D., 3rd Vice Pres.....*Miami*
SAMUEL M. DAY, M.D., Sec'y-Treas.....*Jacksonville*
SHALER RICHARDSON, M.D., Editor.....*Jacksonville*

MANAGING DIRECTOR

STEWART G. THOMPSON, D.P.H.....*Jacksonville*
ERNEST R. GIBSON, Assistant.....*Jacksonville*

BOARD OF GOVERNORS*

DAVID R. MURPHEY, JR., M.D., Chm...(Ex Officio) *Tampa*
WILLIAM M. ROWLETT, M.D...AL-52.....*Tampa*
JOHN D. MILTON, M.D...D-52.....*Miami*
DUNCAN T. MCEWAN, M.D...B-53.....*Orlando*
JAMES H. POUND, M.D...A-54.....*Tallahassee*
NORVAL M. MARR, M.D...C-55.....*St. Petersburg*
WALTER C. PAYNE, M.D...PP-52.....*Pensacola*
HERBERT E. WHITE, M.D...PP-53.....*St. Augustine*
ROBERT B. MCIVER, M.D. (Ex Officio).....*Jacksonville*
SAMUEL M. DAY, M.D. (Ex Officio).....*Jacksonville*
HERBERT L. BRYANS, M.D...S.B.H.-52.....*Pensacola*
STEWART G. THOMPSON, D.P.H. (Advisory)..*Jacksonville*

*SUB-COMMITTEES

1. Veterans Care
FREDERICK H. BOWEN, M.D., Chm.....*Jacksonville*
A. JUDSON GRAVES, M.D.....*Jacksonville*
DOUGLAS D. MARTIN, M.D.....*Tampa*
EDWARD F. FOX, M.D.....*Miami*

2. Liaison — National Foundation Infantile Paralysis
FREDERICK H. BOWEN, M.D., Chm.....*Jacksonville*
A. JUDSON GRAVES, M.D.....*Jacksonville*
H. PHILLIP HAMPTON, M.D.....*Tampa*
EDWARD F. FOX, M.D.....*Miami*

3. Review of Fee Schedules
JOHN D. MILTON, M.D., Chm.....*Miami*
THOMAS H. BATES, M.D.....*Lake City*
FREDERICK H. BOWEN, M.D.....*Jacksonville*
J. BROWN FARRIOR, M.D.....*Tampa*
JOSEPH C. HAYWARD, M.D.....*Orlando*
FLOYD K. HURT, M.D.....*Jacksonville*
IRWIN S. LEINBACH, M.D.....*St. Petersburg*
HAROLD G. NIX, M.D.....*Tampa*
JACK O. W. RASH, M.D.....*Miami*
KENNETH S. WHITMER, M.D.....*Miami*
ADELBERT F. SCHIRMER, M.D.....*Orlando*

4. Blue Shield
LEIGH F. ROBINSON, M.D., Chm.....*Ft. Lauderdale*

SCIENTIFIC WORK

JAMES N. PATTERSON, M.D., Chm...AL-52.....*Tampa*
JAMES L. BORLAND, M.D...B-52.....*Jacksonville*
CAROL C. WEBB, M.D...A-53.....*Pensacola*
FREDERICK K. HERPEL, M.D...D-54.....*W. Palm Beach*
JERE W. ANNIS, M.D...C-55.....*Lakeland*

LEGISLATION AND PUBLIC POLICY

EUGENE G. PEEK, SR., M.D., Chm...B-54.....*Ocala*
J. MAXEY DELL, JR., M.D...AL-52.....*Gainesville*
RUSSELL B. CARSON, M.D...D-52.....*Ft. Lauderdale*
DANIEL A. MCKINNON, M.D...A-53.....*Marianna*
ANGUS D. GRACE, M.D...C-55.....*Fort Myers*
DAVID R. MURPHEY, JR., M.D. (Ex Officio).....*Tampa*
SAMUEL M. DAY, M.D. (Ex Officio).....*Jacksonville*

MEDICAL EDUCATION AND HOSPITALS*

JULIUS C. DAVIS, M.D., Chm...A-53.....*Quincy*
JOSHUA C. DICKINSON, M.D...AL-52.....*Tampa*
THOMAS C. KENASTON, M.D...B-52.....*Cocoa*
ELBERT MCLAURY, M.D...D-54.....*Hollywood*
S. CARNES HARVARD, M.D...C-55.....*Brooksville*

*SPECIAL ASSIGNMENT

1. Urge Gifts to American Medical Education Foundation

PUBLIC RELATIONS*

EUGENE B. MAXWELL, M.D., Chm...AL-52.....*Tampa*
FRANCIS T. HOLLAND, M.D...A-52.....*Tallahassee*
LEIGH F. ROBINSON, M.D...D-53.....*Ft. Lauderdale*
HOWARD V. WEEMS, SR., M.D...C-54.....*Sebring*
EUGENE G. PEEK, JR., M.D...B-55.....*Ocala*

*SPECIAL ASSIGNMENT

1. Rural Educational Program
2. State Education Campaign

NECROLOGY

JOSEPH HALTON, M.D., Chm...C-53.....*Sarasota*
THEODORE C. KERAMIDAS, M.D...AL-52....*Winter Haven*
CHARLES H. DAFFIN, M.D...A-52.....*Panama City*
WALLACE H. MITCHELL, M.D...D-54.....*Key West*
JOSEPH J. LOWENTHAL, M.D...B-55.....*Jacksonville*

MEDICAL POSTGRADUATE COURSE

TURNER Z. CASON, M.D., Chm...B-55.....*Jacksonville*
ROBERT J. PATTERSON, M.D...AL-52.....*Hollywood*
WILLIAM C. ROBERTS, M.D...A-52.....*Panama City*
FRANZ H. STEWART, M.D...D-53.....*Miami*
J. BROWN FARRIOR, M.D...C-54.....*Tampa*

CANCER CONTROL

FRAZIER J. PAYTON, M.D., Chm...D-54.....*Miami*
C. FRANK CHUNN, M.D...AL-52.....*Tampa*
HAROLD O. BROWN, M.D...C-52.....*Tampa*
ROBERT L. TOLLE, M.D...B-53.....*Orlando*
GEORGE W. MORSE, M.D...A-55.....*Pensacola*

MEDICAL ECONOMICS

MERRITT R. CLEMENTS, M.D., Chm...A-52....*Tallahassee*
EDWARD R. ANNIS, M.D...AL-52.....*Miami*
JOHN E. MAINES, JR., M.D...B-53.....*Gainesville*
HARRISON A. WALKER, M.D...D-54.....*Miami*
WILLIAM H. WALTERS, JR., M.D...C-55.....*Lacoochee*

VENEREAL DISEASE CONTROL

FRANK J. PYLE, M.D., Chm...B-52.....*Orlando*
JULIO J. GUERRA, M.D...AL-52.....*Clearwater*
JOHN C. MCSWEEN, JR., M.D...A-53.....*Pensacola*
WILEY M. SAMS, M.D...D-54.....*Miami*
MELVIN M. SIMMONS, M.D...C-55.....*Sarasota*

INTERRELATIONSHIP

HENRY J. PEAVY, SR., M.D., Chm...D-54...*Ft. Lauderdale*
C. MCK. TYRE, M.D...AL-52.....*Eustis*
J. POWELL ADAMS, M.D...A-52.....*Panama City*
ORVILLE L. BARKS, M.D...B-53.....*Sanford*
EMMETT E. MARTIN, M.D...C-55.....*Haines City*

TUBERCULOSIS AND PUBLIC HEALTH*

ALVIN L. STEBBINS, M.D., Chm...A-52.....*Pensacola*
V. MARKLIN JOHNSON, M.D...AL-52....*West Palm Beach*
PHILLIP W. HORN, M.D...B-53.....*Jacksonville*
ERASMUS B. HARDEE, M.D...D-54.....*Vero Beach*
LOUIS J. GARCIA, M.D...C-55.....*Tampa*

*SPECIAL ASSIGNMENT

1. Diabetes Control

STATE CONTROLLED MEDICAL INSTITUTIONS

William D. Rogers, M.D., Chm...A-52...*Chattahoochee*
Gordon H. McSwain, M.D...AL-52.....*Arcadia*
Bailey B. Sory, Jr., M.D...D-53.....*Palm Beach*
James G. Lyerly, M.D...B-54.....*Jacksonville*
Samuel G. Hibbs, M.D...C-55.....*Tampa*

MATERNAL WELFARE

E. Frank McCall, M.D., Chm...B-52.....*Jacksonville*
William G. Meriwether, M.D...AL-52.....*Plant City*
Lowrie W. Blake, M.D...C-53.....*Bradenton*
John N. Sims, Sr., M.D...A-54.....*Live Oak*
Ralph W. Jack, M.D...D-55.....*Miami*

CHILD HEALTH

Egbert V. Anderson, M.D., Chm...A-52.....*Pensacola*
Fred S. Gachet, M.D...AL-52.....*Lakeland*
Luther W. Holloway, M.D...B-53.....*Jacksonville*
Warren W. Quillian, M.D...D-54.....*Coral Gables*
Daniel F. H. Murphey, M.D...C-55.....*St. Petersburg*

CONSERVATION OF VISION

R. Renfro Duke, M.D., Chm...AL-52.....*Tampa*
Nathan S. Rubin, M.D...A-52.....*Pensacola*
William Y. Sayad, M.D...D-53.....*West Palm Beach*
Sherman B. Forbes, M.D...C-54.....*Tampa*
G. Tayloe Gwathmey, M.D...B-55.....*Orlando*

ADVISORY TO WOMAN'S AUXILIARY

C. Robert DeArmas, M.D., Chm...B-55...*Daytona Beach*
M. Eldridge Black, M.D...AL-52.....*Clearwater*
J. Lloyd Massey, M.D...A-52.....*Quincy*
Edward F. Shaver, M.D...C-53.....*Tampa*
James L. Anderson, M.D...D-54.....*Miami*

REPRESENTATIVES TO INDUSTRIAL COUNCIL*

Charles R. Burbacher, M.D., Chm...D-55..*Coral Gables*
Herschel G. Cole, M.D...AL-52.....*Tampa*
Julius C. Davis, M.D...A-52.....*Quincy*
G. Frederick Ortjen, M.D...B-53.....*Jacksonville*
Chas. L. Farrington, M.D...C-54.....*St. Petersburg*

*SPECIAL ASSIGNMENT

1. *Industrial Health*

COUNCILOR DISTRICTS AND COUNCIL

William C. Roberts, M.D., Chm...AL-52...*Panama City*
First—Arthur J. Butt, M.D...1-52.....*Pensacola*
Second—Benjamin A. Wilkinson, M.D...2-53.*Tallahassee*
Third—Eugene G. Peek, Jr., M.D...3-52.....*Ocala*
Fourth—Eugene L. Jewett, M.D...4-53.....*Orlando*
Fifth—Hugh G. Reaves, M.D...5-53.....*Sarasota*
Sixth—Leldon W. Martin, M.D...6-52.....*Sebring*
Seventh—Adrian M. Sample, M.D...7-52....*Fort Pierce*
Eighth—Donald W. Smith, M.D...8-53.....*Miami*

GRIEVANCE COMMITTEE

Walter C. Payne, M.D., Chm.....*Pensacola*
Herbert E. White, M.D.....*St. Augustine*
Joseph S. Stewart, M.D.....*Miami*
William C. Thomas, Sr., M.D.....*Gainesville*
Shaler Richardson, M.D.....*Jacksonville*

ADVISORY TO SELECTIVE SERVICE FOR PHYSICIANS AND ALLIED SPECIALISTS

J. Rocher Chappell, M.D., Chm.....*Orlando*
Thomas H. Bates, M.D..."A".....*Lake City*
Frank L. Fort, M.D..."B".....*Jacksonville*
Alvin L. Mills, M.D..."C".....*St. Petersburg*
John D. Milton, M.D..."D".....*Miami*

EMERGENCY MEDICAL SERVICE

James L. Borland, M.D., Chm.....*Jacksonville*
Merritt R. Clements, M.D..."A".....*Tallahassee*
Vernon A. Lockwood, M.D..."B".....*St. Augustine*
Herschel G. Cole, M.D..."C".....*Tampa*
Frederick K. Herpel, M.D..."D".....*West Palm Beach*

A.M.A. HOUSE OF DELEGATES

Homer L. Pearson, Jr., M.D., Delegate.....*Miami*
Frank D. Gray, M.D., Alternate.....*Orlando*
(Terms expire Dec. 31, 1952)
Louis M. Orr, II, M.D., Delegate.....*Orlando*
Joshua C. Dickinson, M.D., Alternate.....*Tampa*
(Terms expire Dec. 31, 1953)
Herbert L. Bryans, M.D., Delegate.....*Pensacola*
(Term expires Dec. 31, 1952)

BOARD OF PAST PRESIDENTS

William E. Ross, M.D., 1919.....*Jacksonville*
H. Marshall Taylor, M.D., 1923.....*Jacksonville*
John C. Vinson, M.D., 1924.....*Tampa*
John S. McEwan, M.D., 1925.....*Orlando*
H. Mason Smith, M.D., 1926.....*Tampa*
John A. Simmons, M.D., 1927.....*Arcadia*
Frederick J. Waas, M.D., 1928.....*Jacksonville*
Julius C. Davis, M.D., Chm., 1930.....*Quincy*
William M. Rowlett, M.D., 1933.....*Tampa*
Homer L. Pearson, Jr., M.D...1934.....*Miami*
Herbert L. Bryans, M.D., 1935.....*Pensacola*
Orion O. Feaster, M.D., 1936.....*Renton, Wash.*
Edward Jelks, M.D., 1937.....*Jacksonville*
W. Henry Spiers, M.D., 1938.....*West Palm Beach*
Leigh F. Robinson, M.D., 1939.....*Ft. Lauderdale*
Walter C. Jones, M.D., 1941.....*Miami*
Eugene G. Peek, Sr., M.D. 1943.....*Ocala*
John R. Boling, M.D., 1944, 1945.....*Tampa*
Shaler Richardson, M.D., 1946.....*Jacksonville*
William C. Thomas, Sr., M.D., 1947.....*Gainesville*
Joseph S. Stewart, M.D., 1948.....*Miami*
Walter C. Payne, M.D., 1949.....*Pensacola*
Herbert E. White, M.D., Sec'y., 1950.....*St. Augustine*

VOLUME XXXVIII
NUMBER 10

The Journal of the Florida Medical Association

OWNED AND PUBLISHED BY FLORIDA MEDICAL ASSOCIATION

P. O. BOX 1018 JACKSONVILLE, FLORIDA

Editor
Shaler Richardson, M.D.

Assistant Editor
Webster Merritt, M.D.

Associate Editors

Louis M. Orr, II, M.D. ..... Orlando
Franz H. Stewart, M.D. ..... Miami
James R. Boulware, Jr., M.D. ..... Lakeland
Herschel G. Cole, M.D. ..... Tampa
Wilson T. Sowder, M.D. ..... Jacksonville
Carlos P. Lamar, M.D. ..... Miami
Walter C. Payne, M.D. ..... Pensacola

Managing Editor
Stewart G. Thompson, D.P.H.

Committee on Publication

Shaler Richardson, M.D., Chairman ..... Jacksonville
Chas. J. Collins, M.D. ..... Orlando
James N. Patterson, M.D. ..... Tampa

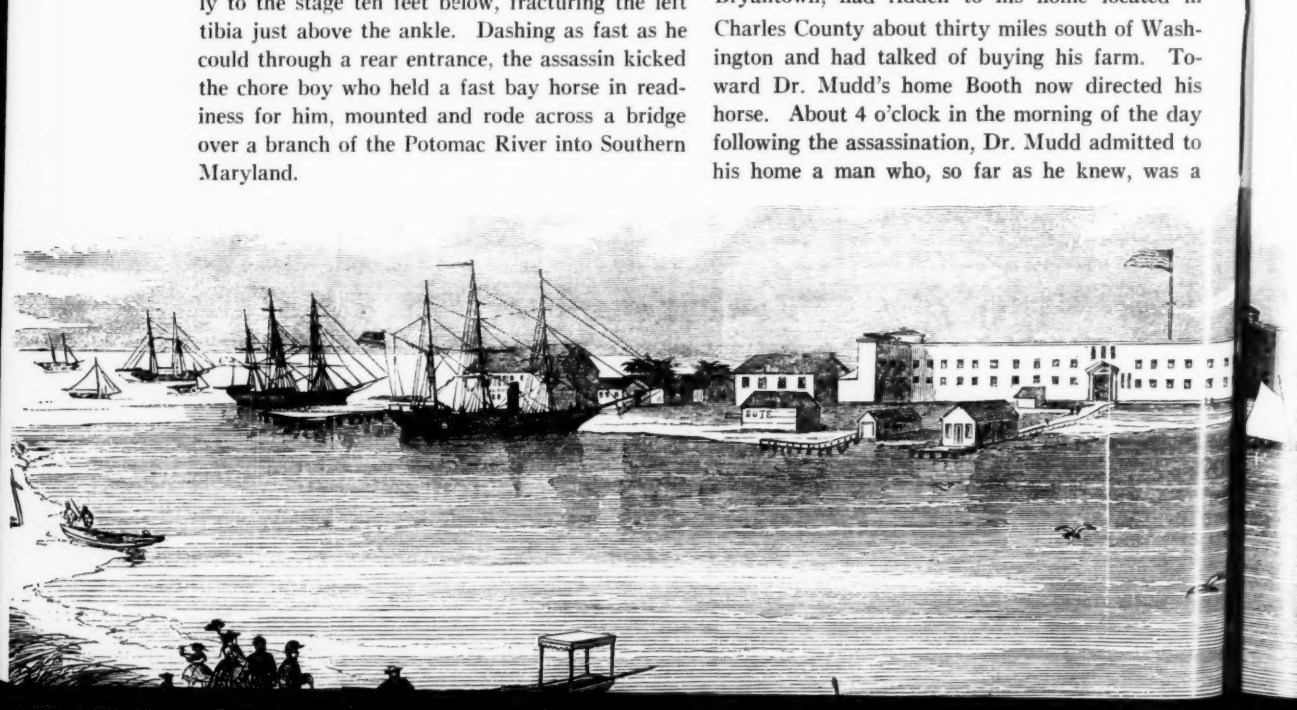
Abstract Department

Kenneth A. Morris, M.D., Chairman ..... Jacksonville
Walter C. Jones, M.D. ..... Miami

His Name Was Mudd

After John Wilkes Booth shot Abraham Lincoln at point blank range in the President's box at Ford's Theater in Washington, D. C., on the evening of April 14, 1865, he leaped from the box, caught a spur of one riding boot on the United States flag which draped the box and fell awkwardly to the stage ten feet below, fracturing the left tibia just above the ankle. Dashing as fast as he could through a rear entrance, the assassin kicked the chore boy who held a fast bay horse in readiness for him, mounted and rode across a bridge over a branch of the Potomac River into Southern Maryland.

Although Booth's plans had been laid carefully, he had of course not reckoned with an accident which would fracture his leg. As he rode south, he must have recalled that during the preceding November he had met Dr. Samuel A. Mudd near Bryantown, had ridden to his home located in Charles County about thirty miles south of Washington and had talked of buying his farm. Toward Dr. Mudd's home Booth now directed his horse. About 4 o'clock in the morning of the day following the assassination, Dr. Mudd admitted to his home a man who, so far as he knew, was a



stranger to him, for his features were disguised. With the stranger was a young man of boyish appearance who later proved to be David Herold. Both gave assumed names.

After the leg had been set and bandaged by the doctor, the stranger rested while his young accomplice went in search of a carriage. None being available, the two men decided to depart on horseback in the early afternoon during the doctor's absence. As they descended the stairs, Mrs. Mudd's suspicions were aroused when the crippled man's false whiskers became partially detached from his face. Upon the return of Dr. Mudd, his wife told him of her suspicions. The following day, from church in Bryantown, Dr. Mudd sent a message to the authorities telling of his experiences with the suspicious strangers. A few days later, he gave to an investigating officer the boot which he had cut off the patient's foot, inside of which was written "J. Wilkes ——." This boot furnished evidence which permitted the authorities to conclude one week after that tragic night that Booth was the assassin.

The violent death of Abraham Lincoln probably shocked the world as much as any previous single event in all history. At a time when better days seemed to be dawning for the nation as a whole, the assassination aroused throughout the land, North and South, an unparalleled excitement. Lincoln had become almost deified in the minds of Northern people, and many in the South were beginning to understand and respect him for his lofty virtues as a man.

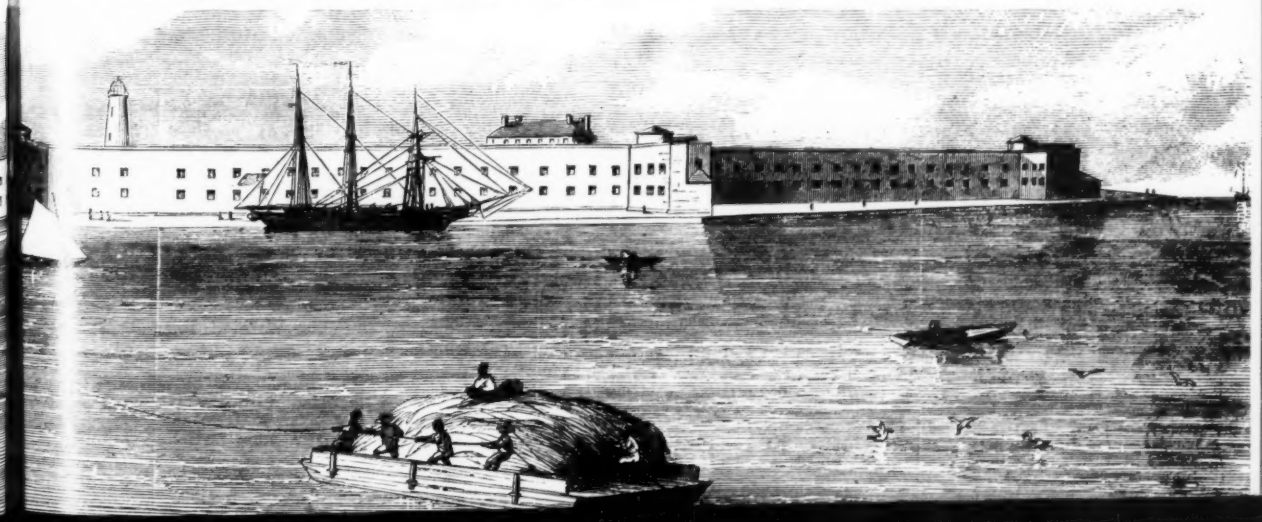
Ralph Waldo Emerson said that the gloom of the calamity had traveled over sea and land from country to country "like the shadow of an uncalculated eclipse over the planet." Emerson doubted whether any one death had ever caused so much pain to mankind. Queen Victoria wrote a personal note to Mrs. Lincoln. Said Disraeli, England's Prime Minister, "There is in the character of the victim. . .something so homely and innocent. . . [that] touches the heart of nations. . . ." At first, General Robert E. Lee refused to hear the details of the assassination. The General said that he regretted the death of Mr. Lincoln as much as any man in the North and believed him to be the epitome of magnanimity and good faith.

Among those high in authority, however, some were at variance with the spirit of magnanimity and kindness of Lincoln; they developed a deep-seated conviction that victims must be offered for sacrifice. Dr. Mudd became one of these victims.

Born in Charles County, Maryland, on Dec. 20, 1833, Samuel A. Mudd became proficient in Greek, Latin and French and also in music, performing with skill on the violin, piano and flute. He was graduated from Georgetown College and then from the University of Maryland Medical School in 1856. After a one year internship, he began the practice of medicine on his father's large estate in Charles County. About a year and a half later, he married Miss Sarah Frances Dyer, whom he addressed later in many letters as "My darling Frank." He was a kindly country practitioner, who enjoyed nothing more than a peaceful hour with his family.

On April 26, Booth was shot and killed near Port Royal, Va., in a barn which had been set on fire and surrounded. Dr. Mudd and seven others were arrested and charged with complicity in the crime. At first Dr. Mudd did not take his arrest very seriously. Little did he realize that it would

Sketch of Fort Jefferson, Garden Key, Tortugas Islands, Florida, "The Key to the Gulf of Mexico," about the time Dr. Mudd was imprisoned there. Library of one of the Editors.



be four years before he would see his loved ones and home again.

President Andrew Johnson ordered on May 1 that the accused be tried by nine military officers. Many eminent attorneys throughout the country, including General Thomas Ewing who ably defended Dr. Mudd, contended that the civil courts alone had jurisdiction to try the accused.

The trial, which began on May 10 and ended on June 30, is thought by many to have been the worst blot on the American record of justice. Daniel Thomas, an outcast from his home who years later died in the penitentiary after being convicted of pension frauds, apparently gave false testimony against Dr. Mudd in the hope that he would receive part of the large reward offered for the capture and conviction of Booth and his alleged accomplices. Four of the accused were sentenced to be executed. One was sentenced to be confined at hard labor for six months. Dr. Mudd and two others were sentenced to be confined at hard labor for life.

On July 24, Dr. Mudd entered Fort Jefferson to begin his sentence. Construction of the fort had been started in 1846, on Garden Key in the Dry Tortugas group, some seventy miles west of the city of Key West. The structure was intended to complete a great link of forts from Maine to Texas. During construction the fort began to sink and crack, for the island was not solid rock. Although a shot was never fired from its cannon, the fort went down in history as one of the worst prisons and "death holes" anywhere in the world. First laborers, then soldiers and finally prisoners were victims of disease and hurricanes which swept the area.

In a letter addressed to his wife on August 24, Dr. Mudd stated that he was at that time serving in the hospital and had no labor to perform. His food consisted of coffee, bread and butter three times daily; occasionally Irish potatoes; molasses when he was able to buy it; fish when they could be caught.

In September, Dr. Mudd unfortunately attempted to escape and when captured was put at hard labor. Upon the arrival of every incoming ship, he was placed in the dungeon. On September 30, he wrote that at least three fourths of the inmates had suffered from "bone fever" (dengue). In October, he wrote that he was confined to a small damp room. In November, he told how the prisoners had been placed in leg irons without cause, marched to headquarters and put at hard labor cleaning old brick. In December, he stated: "We are still in irons. . . ." In January 1866:

> I have grown old in my youth. . . .Imagine one loaded down with heavy chains, locked up in a wet, damp room twelve hours out of every twenty-four on working days and all day on Sundays and holidays. . . . The atmosphere we breathe is highly impregnated with sulphuric hydrogen gas. . .generated by the numerous sinks that empty into that portion of the sea. . .which is immediately under a small port hole — the only admission for air and light we have from the external port. My legs and ankles are swollen and sore. . . .My hair began falling out sometime ago. . . .My eyesight is beginning to grow very bad, so much so that I can't read or write by candlelight. . . .My eyes are painful and irritated. . . .With all this imagine my gait with a bucket and broom, and a guard. . . .This has been our treatment for the last three months coupled with bad diet, bad water, and every inconvenience. . . ."

In July 1866, General Phil Sheridan intervened and secured better treatment for the prisoners. In August of that year, Dr. Mudd wrote that he was enjoying better health, which he attributed to the kindness of the officials in giving them a plank floor for their sleeping quarters.

On Aug. 18, 1867, a severe epidemic of yellow fever began at Fort Jefferson. When the post surgeon was stricken and was among the first to die, Dr. Mudd was placed in charge of the local hospital. One of his first acts was to abolish a hospital on distant Sand Key, to which "sick patients were conveyed seated in small boats facing coffins piled up in the bow." It is recorded that in many instances coffins were placed alongside the hospital beds there, in readiness to receive the body of patients expected to die, and "no sooner had the breath left the body, but that it was coffined and hurried over to its last resting place."

On September 8, Dr. Mudd wrote of the situation on Garden Key: "Nearly every man now on the island is infected with the disease. . . .The greatest consternation prevails. . . ." On September 13, he wrote: "Although three fourths of the garrison have been removed, the epidemic seems to increase with unabated fury. . . .I remain up every night until eleven or twelve, and sometimes later. . . ." The most remarkable spread occurred on the night of September 16 when thirty-five men composing one half of company M were stricken with the most malignant form of the disease between 11 and 2 o'clock.

On October 4, Dr. Mudd was stricken with the fever, but two weeks later he wrote that he had recovered. An unsigned petition was prepared by the garrison praising his work and requesting his release. After the epidemic subsided, however, he was again put in chains.

Finally, as a result of the unceasing efforts of

his wife and friends, Dr. Mudd was pardoned by President Johnson. He regained his freedom on March 8, 1869, after being imprisoned at Fort Jefferson for almost four years.

Although Dr. Mudd returned to Maryland and lived for nearly thirteen years, he apparently never again enjoyed robust health. While serving the sick at nighttime during a winter storm, he contracted pneumonia. Nine days later, on Jan. 10, 1883, he died. A martyr, not yet fifty years of age, Dr. Mudd was faithful to his patients and to his profession until the very end.

Dr. Samuel A. Mudd

From "The Life of Dr. Samuel A. Mudd" by Nettie Mudd, New York, The Neale Publishing Company, 1906. Jacksonville Public Library.

"Doctor, What Plans Have You Made?"

Are you looking ahead, Doctor? Have you given careful thought to intelligent financial plans for the future? "But physicians are, perhaps, the poorest examples of constructive planning." So wrote John Alan Appleman, J.D., in a thought-provoking article entitled "Even Physicians Die," which was published in the December 15 issue of the Journal of the American Medical Association. Every member of the medical profession would do well to peruse it with care.

The picture portrayed of young Dr. Jones as he climbs the ladder of success may cause the reader discomfort, but only so because it strikes too close to home. The author finds it "typical of the majority of physicians — some of whom survive to experience a few of these headaches, others of whom leave these problems to widows and heirs."

Dr. Appleman makes clear the physician's need for estate analysis — a specialty combining elements of taxation, will review, trusts, investments, insurance, and many other phases of law and business, which has come into being because of acute taxation and personal problems such as confronted Dr. Jones. Basically, it is "a philosophy — a philosophy both for living and for death which works to solidify the relationships existing between a man and his family and to provide for the family's comfort after his death, but without destroying them as persons." It is as much concerned, however, with the art of successful living as it is with plans for death.

Physicians who missed this article will profit by digging out the Dec. 15, 1951 A.M.A. Journal and reading it, as did one Journal reader who thoughtfully directed the attention of the Editors to it.

American Medical Education Foundation

April 1 marks the beginning of the 1952 drive of the American Medical Education Foundation for funds to continue the work of supporting medical education. "Though much was done in '51 — there's more to do in '52," said Dr. Elmer L. Henderson, Louisville, Ky., President of the Foundation, as he sounded the call to action for the second year's drive at a conference in Chicago in mid-February. Physician leaders in medical philanthropy from forty-eight states attended this meeting, held for the purpose of stimulating action by the individual states for the current drive being conducted during April, May and June. Dr. Jack Q. Cleveland of Coral Gables ably represented the Florida Medical Association at the conference.

In a special message in the February 1952 issue of The Journal, President David R. Murphey, Jr., set forth tersely the need for and the background of this movement which has already gained great

momentum as it enters its second year. Hardly a member of the medical profession is unaware of the precarious financial condition of this country's medical schools today. Most physicians also realize that the American Medical Association forthrightly led a movement to remedy their plight by organizing the American Medical Education Foundation and contributed half a million dollars to serve as a nucleus for last year's fund. It has made a gift this year in like amount, bringing to a total of one million dollars its contributions to the Foundation since its inception little more than a year ago. It also underwrites all of the Foundation's expenses so that every dollar of every contribution received goes in full to the medical schools.

An important change in policy this year makes it possible for alumni and friends of a specific medical school to contribute to that school through the Foundation and the school will receive the full amount of the contribution plus its full share of unearmarked funds.

The first year's experience has demonstrated that the success of the foundation is to be measured in terms of action at the state and county level. The number of contributions already received in 1952, prior to the opening of the campaign, reflects the growing interest largely stimulated by the increasingly active efforts of state and county societies. The Florida Medical Association will undoubtedly wish to have a generous share in this nationwide project.

Many physicians are recognizing for the first time the debt they owe their medical schools, while many others see in the activities of the Foundation a channel through which the medical profession has an excellent opportunity to demonstrate its willingness to aid in the advancement of high standards of medical education. The success of the present effort will constitute a real challenge to business, industry, agriculture and other important groups concerned with medical education to contribute generously to the solution of this problem. The source from which financial support is obtained will inevitably determine whether the medical schools are to continue to function as integral and independent institutions or whether they are to become subject to federal supervision and subsequent control.

Apathy in Voting

"Socialism can easily come to this country without a vote of the people. It can come by default." This warning in the Dallas Morning News not long ago was provoked by information carried in Dallas, Texas, newspapers on the response of the voting public in that city. With only 26 per cent of the members of the seven leading Dallas professional groups voting in the last Presidential election, the heaviest vote was in the legal profession with a mere 38 per cent of the lawyers casting their ballot. The medical profession was a close second to the lawyers with 36 per cent of the physicians voting. The nursing profession made the poorest showing with only 12 per cent.

In the light of such figures it is understandable that the Dallas Morning News should envision socialism by default and comment further:

"It is the doctor, the lawyer, the engineer who bemoan the threat of socialism and burdensome debts. The doctor who fears socialized medicine and does nothing at the polls to combat it is legion. The Dallas poll merely proves locally a national picture that is pitiable.

"The result is a government of pressure groups alert to step in where others fail to tread.

"Polls similar to that in Dallas show 85 per cent of labor union membership votes. The figure is 90 for those who hold government jobs. Not only do they vote, they influence others. This is their civic privilege; we might even say duty, though it is a duty with a motive."

Remarking editorially upon the apathy of citizens of this country regarding voting, the Los Angeles Mirror recently estimated that less than 50 per cent of qualified United States voters exercise their privilege of voting in national elections. The voting rate in Los Angeles is between 20 and 35 per cent, a dismal showing indeed when compared to Great Britain, where national elections are said to bring to the polls 85 per cent of the voters.

Every physician should read the editorial, "Voting Privileges," which appeared in the Dec. 29, 1951 issue of the Journal of the American Medical Association. In showing up the apathy of the citizens when the elections roll around, it summarized the aforementioned newspaper information and expressed the belief that professional groups, especially physicians, now show a much greater interest in voting. "It certainly can be said without fear of contradiction that in the country at

large there has been sharply increased interest in registering and voting on the part of the medical profession. However, there is still room for improvement, and all physicians, as well as other members of professional groups, and especially those concerned with health, should recognize their obligations as citizens in these critical times."

Surely, as suggested, the professional groups should begin now without further delay to shoulder their responsibilities as voters and citizens and to take full advantage of the peculiar opportunity which is theirs for leadership in civic activities. The "year of decision" is at hand, and there is no time to lose. Waiting till the last minute may be too late to better the 1948 record.

New Tests in the Etiologic Diagnosis of Diabetes Mellitus

Is the "Higher Cost of Medical Care" Justified?

The technologic advances of the past two decades have introduced radically new factors in the practice of medicine. These new factors have so increased the complexity in the field of medical diagnosis and in the interpretation of the mechanisms of disease that the amount of knowledge and time for study and the cost of the equipment required of the average physician today can no longer be compared with those required of the practitioner of the first quarter of this century.

Each new technologic discovery has opened a vast new field of research. Each new research activity has added to the complexity of medical science and, as a natural corollary, to the economic burden of an up-to-date type of medical practice. The stethoscope, the microscope, the x-ray machine, the electrocardiograph, the spectrophotometer, are but a few of the innumerable milestones on the road to progress in the care of the sick and in the prevention of disease. Each one of them, of course, has added an ever increasingly expensive item to the bill we must pay for medical care.

Are these ever growing costs worth while to us as members of the human race? How do they reflect upon our everyday living as individuals, and as a nation?

One answer is given in the Statistical Bulletin of the Metropolitan Life Insurance Company for January 1952.[1] The industrial policyholders of this company, who constitute a large cross section of wage earners and their families in the United States and Canada, have shown such an improvement in their health that the average length of life in this group has increased by five years in the past decade and by ten years since the early 1930's, even with the the inclusion of war deaths in the computations. "Under the mortality conditions of 1911, the expectation of life at birth in this insured group was 46.6 years, or 6½ years less than for the general population of the U.S." In 1951 this life expectancy has reached the new high of 68.5 years, an extension of 21.9 years during the last four decades. "The very favorable health conditions which now prevail reflect the cumulative effect of marked advances in all branches of medical science and practice, the growth and the more efficient use of medical and public health facilities and the economic prosperity of recent years," which have made such advances and their application possible.

As an illustration of the influence of these costlier but so useful technologic improvements, we find that "Diabetes, one of the major degenerative diseases, registered a decline of five per cent in the crude death rate from 1950 to 1951."

Mortality rates in diabetes have been declining for thirty years, since the discovery and application of insulin has permitted better understanding and management of the disease. But science is never satisfied at any level of progress. Further improvements are constantly sought. Research continues, stimulated by newer discoveries in related fields of science. Now we are able further to clear up the fog of ignorance, and as we become better acquainted with the intimate mechanisms of carbohydrate metabolism, we may expect an even greater degree of diabetes control. The results are already showing in a rapidly expanding life expectancy and a better life for the diabetic patient.

In the past decade it has been emphasized that normal carbohydrate tolerance depends in many instances not on the functional integrity of the pancreas but rather on a normally functioning liver and on maintenance of the balance between the action of insulin and the hormones of the anterior lobe of the pituitary and the adrenal cortex. Translated into simple facts, these observations mean that we know diabetes mellitus is a syndrome which may be produced by a variety of pathogenic mechanisms. The pancreas, which for many years was considered the seat of the primary disturbance, may even remain entirely normal, both in the organic and the functional senses.

A purely hepatic diabetes, nonendocrine and due primarily to disease of the liver, can now be demonstrated. Endocrine types of diabetes may

be produced by disturbances of either the pancreas or the pituitary-adrenal axis.

As a result of pituitary-adrenocortical stimulation with an increase in circulating ll-oxysteroids, the administration of glucose in the normal person is followed immediately by a fall in the absolute lymphocyte count. This phenomenon occurs only in about one half of all diabetic patients, indicating a normally functioning pituitary-adrenal axis. The other half probably suffers some disturbance of this axis, so that the lymphocyte response to administered glucose may be employed to distinguish a diabetes associated with dysfunction of the pituitary-adrenal axis from one due to other factors.

There is also a decrease in serum inorganic phosphorus in the normal person after glucose administration. This phenomenon is not seen in a large percentage of diabetic patients. Since experimentally the response of serum inorganic phosphorus level occurs in the absence of the liver but not in the pancreatectomized animal, this reaction is believed to be significant of peripheral glucose utilization and has been suggested by Forsham and Thorn to distinguish diabetes associated with normal peripheral glucose utilization (intact pancreas), assumed to be due to hepatic dysfunction, from diabetes associated with deficient peripheral glucose utilization, considered to be due to either relative (pituitary-adrenal) or absolute (pancreatic) insulin lack.

In a recent study by Lazarus and his co-workers,[2] the simultaneous study of the response of lymphocyte counts and phosphorus levels to administration of glucose in 17 unclassified cases of known diabetes helped to separate them into 9 cases in which the etiology was probably pituitary-adrenal dysfunction, 6 in which diabetes was due to hepatic dysfunction and only 2 in which the disease was actually due to pancreatic insufficiency.

These findings will not necessarily reflect immediately upon our present methods of treatment for the diabetic patient. The time-proved adequate diet, insulin and exercise are still the basis of correct management of the disease. But who can deny that, as we begin to differentiate the disease into more accurate etiologic classifications, our methods of control will improve? In view of these recent technologic improvements, it would not be too adventurous to predict the possibility of an eventual permanent cure for at least certain types of diabetes.

These considerations seem to justify fully the expense of new diagnostic technics. We must accept the fact that in the present advanced status of our scientific knowledge, a "two dollar diagnosis" no longer will always satisfy completely the needs of our enlightened population.

But these facts must be explained to the lay public if we are to dispel the common criticisms against the high cost of medical care. John Q. Citizen must realize that what he pays for today is better health and extra years of life to enjoy it. No expense can then be considered too high.

1. Excellent Health Record in 1951, Statist. Bull. Metrop. Life Insur. Co. 33:1-7 (Jan.) 1952.
2. Lazarus, S. S.; Volk, B. W.; Jacobi, M., and Gilady, Miriam Y.: Absolute Lymphocyte Count and Serum in Organic Phosphorus after Glucose Administration in Diabetic Patients, Am. J. Clin. Path. 22:127-134 (Feb.) 1952.

Graduate Medical Education

This is the twentieth year the Florida Medical Association and the University of Florida have sponsored graduate medical education for the physicians of Florida. The major feature annually is the Graduate Short Course, which will be presented this year the week of June 23. For approximately ten years the Committee on Medical Postgraduate Course has planned highly specialized courses either the first two days of the Short Course or just prior to the Short Course. Because many wish to attend both the specialized courses and the Short Course, it was decided the special courses should be given the week before the Short Course.

Seminar on Diagnosis

On June 19-21 a Seminar on Diagnosis is being offered at the George Washington Hotel in Jacksonville. The program is a most excellent one, and the specialists, Drs. Lewis M. Hurxthal, Hugh F. Hare and William A. Meissner of the Lahey Clinic, Boston, are all outstanding medical teachers. We are most fortunate to have come to us three such men who are associated in their daily work and who can correlate their lectures. While the program is primarily designed for internists and general practitioners, nevertheless because diagnosis

NOTICE

Your March Journal carried a complete program and other detailed information relative to the Seventy-Eighth Annual Meeting of the Association in Hollywood, April 27-30.

is pertinent to all physicians practicing medicine, we believe any physician would profit by attending. The program follows:

Thursday, 9:30 a.m.

Symposium on Thyroid Disease

Normal Thyroid Function
Simple and Nodular Goiters
(Intermission, 30 minutes)
Intrathoracic Goiter
Thyroid Tumors, Pathology, Diagnosis and Treatment

Thursday, 2 p.m.

Symposium on Thyroid Disease

Physiology and Pathology of Hypofunction
Pituitary Relationships
(Intermission, 15 minutes)
Cretinism, Myxedema
Physiology and Pathology of Hyperfunction, Adenoma, Adenomatous and Hyperplastic Goiter
(Intermission, 15 minutes)
Diagnosis and Treatment

Friday, 9 a.m.

Adrenal Diseases—Dr. Hurxthal, Dr. Meissner

Physiology, Etiology, Diagnosis, Pathology and Treatment (90 minutes)
(Intermission, 15 minutes)

Lymph Node Disease—Dr. Hare, Dr. Meissner, Dr. Hurxthal

Clinical Manifestations, Diagnosis, Pathology and Treatment

Friday, 2 p.m.

Lung Tumors—Dr. Hare, Dr. Meissner

Clinical Manifestations, Diagnosis, Pathology and Treatment (90 minutes)
(Intermission, 15 minutes)

Metabolic Bone Disease—Dr. Hurxthal, Dr. Hare, Dr. Meissner

Clinical Manifestations, Diagnosis, Roentgen and Pathologic Recognition, and Treatment (90 minutes)

Saturday, 9 a.m.

Clinicopathologic Conference, with Discussion of Clinical and Laboratory Methods of Diagnosis

Demonstration (2 cases) — Dr. Hurxthal, Dr. Hare, Dr. Meissner (120 minutes)

Medical Licenses Granted

Dr. Homer L. Pearson, Jr., Secretary of the State Board of Medical Examiners, has reported that of the 190 applicants who took the examination of the Board, held November 26 and 27, 1951 in Jacksonville, 161 passed and have been issued licenses to practice medicine in Florida. The names and addresses of the 161 successful applicants follow:

Abrahamson, Ira Adolph, Jr., Cincinnati, Ohio (U. of Cincinnati 1948)
Adams, Leslie Rudolph, Jacksonville (U. of Pennsylvania 1951)
Akes, Charles Delbert, Tampa (U. of Illinois 1951)
Altman, Donald H., Miami (U. of Tennessee 1950)
Anderson, Raymond Thomas, Detroit, Mich. (Wayne U. 1942)
Anlage, Henry Joseph, Miami (St. Louis U. 1950)

Baker, Hillier Locke, Chicago, Ill. (Rush Med. Coll. 1915)
Barland, Samuel, Elmhurst, N. Y. (Maryland Med. Coll. 1929)
Beidleman, Barkley, Pensacola (Harvard U. 1944)
Belyeu, Jesse Harvey, Jacksonville (Tulane U. 1951)
Bidgood, Willis Deanes, Birmingham, Ala. (U. of Pennsylvania 1946)
Blackford, Florence, Miami (Indiana U. 1934)
Blatt, Lester Jay, Coral Gables (New York U. 1943)
Brandon, James Robert, Greenville, S. C. (Med. Coll. of Virginia 1942)
Braunstein, John Rutley, Cincinnati, Ohio (U. of Cincinnati 1943)
Buerk, Minerva Smith, Evanston, Ill. (Woman's Med. Coll. of Pennsylvania 1946)
Bumgardner, Heath Denton, Philadelphia, Pa. (Temple U. 1942)
Burgh, Lois Cleone, Tallahassee, (Scotland, 1942)
Burke, Sean Kevin, St. Petersburg (Harvard U. 1951)

Campbell, Francis Joseph, Opa Locka (Coll. of P. & S., Boston, 1938)
Capidaglis, Andre Stephan, Miami (Cornell U. 1946)
Carleton, Charles Coleman, Orlando (McGill U. 1950)
Carpenter, Frederick Albertin, Chamblee, Ga. (Emory U. 1951)
Castleberry, Jesse Wendell, Birmingham, Ala. (Northwestern U. 1950)
Cecil, Russell LaFayette, New York, N. Y. (Med. Coll. of Virginia 1906)
Clark, Edward John, Jacksonville (Jefferson 1950)
Coggan, George Michael, Birmingham, Ala. (Med. Coll. of Alabama 1951)
Cole, J. P., Alma, Ark. (Columbia U. 1944)
Conn, James Keener, Staten Island, N. Y. (Washington U. 1948)
Crage, Francis Michael, Chicago, Ill. (U. of Buffalo 1919)
Crago, John Alfred, Rochester, N. Y. (Cornell U. 1950)
Culpepper, Walter Shelley, New Orleans, La. (Tulane U. 1940)

Davis, Courtland Harwell, Jr., Durham, N. C. (U. of Virginia 1944)
Davis, Robert Jay, Lake City (Johns Hopkins U. 1940)
Dayton, Helen Conger, Miami (U. of Tennessee 1951)
Deam, John Henry, Tampa (Jefferson 1951)
Dean, George Walter, Milwaukee, Wis. (U. of Wisconsin 1934)
DeNote, Anthony Paul, Philadelphia, Pa. (Jefferson 1936)
Denser, Clarence Hugh, Chattahoochee (U. of Cincinnati 1917)
Deurloo, Henry William, Gainesville (U. of Michigan 1936)
Duest, Lloyd Joseph, New Bedford, Mass. (Tufts Coll. Med. Sch. 1937)

Eisenbarth, Elmer Jonn, Miami (Indiana U. 1951)
Ettinger, Charles David, Daytona Beach (New York U. 1950)
Eversole, Joseph Wilson, Jacksonville (U. of Virginia 1940)

Feigelson, Howard Harvey, Miami (U. of Cincinnati 1950)
Feigenbaum, David, Miami Beach (Tulane U. 1951)
Flynn, Joseph Charles, Orlando (Jefferson 1951)
Folmer, Edward Joseph, New York, N. Y. (Georgetown U. 1941)
Foster, Richard Leland, Ft. Lauderdale (U. of Louisville 1943)
Freedland, Marvin Sol, Miami (Chicago Med. Sch. 1951)
Fromang, Vernon Leroy, Daytona Beach (Emory U. 1950)

Gaines, Gartrell Jerome, Jr., (Col.), West Palm Beach (Howard U. 1950)
Galluccio, Anthony Carmelon, Ft. Lauderdale (New York U. & Bellvue Med. Coll. 1927)
German, Walter Abel, Jr., Miami (Washington U. 1951)
Gerson, Theodore Frederick, West Palm Beach (Temple U. 1945)
Gillespie, Russell Hardy Dean, Neptune Beach (U. of Michigan 1951)
Gillikin, Charles Monroe, Mobile, Ala. (Bowman Gray Sch. of Med. 1946)
Gillman, Aron Nathan, Coral Gables (U. of Georgia 1945)
Gittelson, George, Miami (U. of Pennsylvania 1951)
Glucroft, Stephen Henry, Jacksonville (Long Island Coll. of Med. 1947)
Goldberger, Emanuel, New York, N. Y. (Long Island Coll. of Med. 1936)
Goodman, Julian John, Ft. Bragg, N. C. (U. of Vermont 1948)
Gouchnour, Thomas Henry, Jacksonville (Indiana U. 1951)
Greenhouse, Jerome Mayer, St. Louis, Mo. (St. Louis U. 1939)
Grosz, Eugene, Evanston, Ill. (Czechoslovakia 1920)
Gunion, Frederick Ashley, Coral Gables (Med. Coll. of Virginia 1951)
Gunsolus, John Martin, Miami (U. of Minnesota 1951)

Hall, Fairfax, New Rochelle, N. Y. (Columbia U. 1911)
Harris, James Allen, Cincinnati, Ohio (Duke U. 1950)
Head, Thomas Duncan, Gainesville (Med. Coll. of Alabama 1950)
Healey, Raymond Walter, Miami (St. Louis U. 1951)
Hobson, John Lewis, Jacksonville (Vanderbilt U. 1949)
Horwitz, Michael Lee, Miami Beach (Chicago Med. Sch. 1951)

Kellerman, Edgar Allen Poe, New York, N. Y. (New York U. 1939)
Kelso, Kip Gordon, Tampa (U. of Oklahoma 1946)
Kobak, Mathew William, Chicago, Ill. (U. of Chicago 1941)
Kulvin, Max Mandel, Miami (Rush Med. Coll. 1916)
Kummer, William Martin, New Orleans, La. (Hahnemann Med. Coll. of Phila. 1941)

LaRoche, Laurent Pierrepont, Orlando (Med. Coll. State of S. C. 1951)
Lawrence, Ralph Lewis, Tallahassee (Med. Coll. State of S. C. 1921)
Leininger, Hilbert Adolph Peter, Miami (Indiana U. 1939)
Levatin, Bernard, Coral Gables (U. of Illinois 1946)
Litt, Edward Theodore, Miami Beach (U. of Pennsylvania 1924)
Lockwood, Raymond Max, Clearwater (U. of Pittsburgh 1943)
Long, Frank Groover, Jacksonville (Temple U. 1951)

Macdonald, Ian John, Orlando (Tufts Coll. Med. Sch. 1951)
Mahoney, Paul Leo Jr., Jacksonville (U. of Arkansas 1951)
Mahrer, Martin Paul, Youngstown, Ohio (Hungary 1915)
McCallen, Albert Merle, Miami (Northwestern U. 1937)
McIntyre, Clifford Edward Locke, South Miami (Columbia U. 1944)
Meadows, Burton Thomas, St. Petersburg (New York U. 1943)
Mein, Robert Malhert, Jacksonville (U. of Louisville 1939)
Meli, John James, Columbus, Ohio (U. of Maryland 1951)
Mendelson, Joel, Panama City (Emory U. 1950)
Menge, Louis, New York, N. Y. (Coll. P. & S., Kansas City 1943)
Mikes, Frank Leo, Shaker Hgts., Ohio (St. Louis U. 1940)
Mitchell, Andrew Chrismond, Tampa (U. of Virginia 1951)
Moncure, William Bigbie, Richmond, Va. (Med. Coll. of Virginia 1949)
Morse, Irwin S., Coral Gables (Chicago Med. Sch. 1950)
Mullen, Sanford Allen, Virginia Beach, Va. (Columbia U. 1949)
Murray, Charles Alvin, Bay Pines (Northwestern U. 1936)

Nash, Norman, Miami (New York Med. Coll. 1948)
Neil, Robert Lewis, Memphis, Tenn. (U. of Tennessee 1951)

O'Brien, Paul Stevens, Miami (Ohio State U. 1951)
Owen, Richard Deen, Tampa (Jefferson 1951)

Parks, Raymond Edward, Miami (Yale U. 1945)
Patras, Mary Cornelia, Miami (Loyola U. 1935)
Patton, William Thomas, New Orleans, La. (Tulane U. 1946)
Pauley, Harvey Chadwick, Jr., St. Petersburg (Tufts Coll. Med. Sch. 1944)
Perdue, Maxine Roberta, Miami (Duke U. 1943)
Perry, John Fleck, Orlando (U. of Buffalo 1951)
Pinkus, Walter Howard, Tampa (U. of Illinois 1947)

Reiger, Anthony C., Miami Beach (Long Island Coll. of Med. 1931)
Robinson, Murry Myer, Washington, D. C. (George Washington U. 1935)
Robertson, James Farish, New Smyrna Beach (U. of Pennsylvania 1913)
Roddy, Stephen Robert, Miami (U. of Tennessee 1951)
Rosof, Jacob Agassiz, Hollywood (U. & Bellevue Med. Coll. 1931)

Schmidt, Ivan Clair, Lantana (Ohio State U. 1943)
Schmitt, Norman Lawrence, Detroit, Mich. (Wayne U. 1936)
Schwarz, Marvin Jerome, Miami Beach (Wayne U. 1950)
Senese, John Gerard, Brooklyn, N. Y. (Long Island Coll. of Med. 1914)
Shackleton, John Henry, Jr., Jacksonville (Jefferson 1943)
Sherman, John James, Huntington, W. Va. (Rush Med. Coll. 1941)
Shupe, Henry Wren, Jacksonville (U. of Oklahoma 1949)
Silberman, Harold Myron, Miami (Johns Hopkins U. 1951)
Sims, Murry, Coral Gables (U. of Michigan 1945)
Skyer, Roslyn, West Palm Beach (Hahnemann Med. Coll. of Phila. 1946)
Skyer, Joseph Robert, West Palm Beach (Hahnemann Med. Coll. of Phila. 1943)
Small, George Allen, Brookline, Mass. (Tufts Coll. Med. Sch. 1936)
Small, Sidney Robert, Los Angeles, Calif. (U. of Illinois 1943)
Smith, Freddie Alphonso (Col.), Tampa (Meharry 1950)
Startzman, John Edward, Orlando (Ohio State U. 1946)
Suarez, George Joseph, Tampa (U. of Havana 1946)
Summers, William Boyd, Miami Beach (U. of Colorado 1951)

Talley, Robert Gene, Miami (U. of Tennessee 1950)
Talley, William Richard, Lake City (U. of Louisville 1942)
Tanner, Charles Louis, Miami (Johns Hopkins U. 1951)
Taylor, Willard Mitchell, Ft. Lauderdale (Indiana U. 1940)
Tierney, Kaden, Hyattsville, Md. (George Washington U. 1942)
Tinkler, William Phillips, Miami (Vanderbilt U. 1951)
Tippett, Samuel Nelson, Miami (Ohio State U. 1951)
Tomlinson, Wray Joseph, Jacksonville (Temple U. 1935)
Turek, Samuel Lester, Chicago, Ill. (Northwestern U. 1935)

Updike, Edwin Hoyt, II, Jacksonville (Columbia U. 1949)

Vihlen, Frederick Englebirth, Miami (Temple U. 1951)

Wainer, William Wolfe, Providence, Ky. (Boston U. 1936)
Walker, Ambrose Taylor, Jr., Mammoth Spring, Ark. (U. of Arkansas 1946)
Walker, Francis Samuel, Orlando (St. Louis U. 1951)
Walker, Nancy Adine, Valparaiso (Louisiana State U. 1949)
Wallace, William Earle, Orlando (Jefferson 1951)
Warrington, James Clyde, Coral Gables (Coll. of P. & S., Boston 1942)
Weir, Hughey Monroe, Jacksonville (Med. Coll. of Alabama 1951)
Wells, Carl Hunt, Jacksonville (George Washington U. 1941)
Wells, Leila Hulbert, Jacksonville (George Washington U. 1941)
White, Charles Maxwell, Miami (U. of Pennsylvania 1951)
Wilber, Stewart Anthony, Jacksonville (Albany Med. Coll. 1946)
Williams, Gerald Stephen, Weirsdale (Tulane U. 1944)
Wilson, Joseph Crawford, Hollandale, Miss. (Tulane U. 1939)
Winslow, Oliver Parry, Jr., Coral Gables (U. of Maryland 1945)
Woodhall, Jerry Philip, Macon, Ga. (Johns Hopkins U. 1941)

Zimmerman, Daniel Harold, Miami Beach (U. of Baffalo 1951)

Procedure for Establishing and Maintaining Cancer Clinics Adopted by Florida Cancer Council, Tampa, Florida, January 18, 1952

Lorenzo L. Parks, M.D., Secretary
Jacksonville

A Cancer Clinic should be located in a general hospital which is approved by the American College of Surgeons.

A general hospital qualified to operate a Cancer Clinic must have the following key staff members available to serve the Cancer Clinic: (1) Pathologist, (2) Radiologist, (3) General Surgeon, (4) Gynecologist, and (5) Internist.

The following specialists should be available from the hospital staff as consultants: (1) Otorhinolaryngologist, (2) Orthopedist, (3) Urologist, (4) Thoracic Surgeon, and (5) Dermatologist.

It is advisable that a standing committee, known as the Cancer Committee or Tumor Board, etc., be appointed by the staff of the hospital. This committee will have the responsibility of the cancer program in the hospital inclusive of the Cancer Clinic.

One of the physician staff members must be designated as the director of the clinic.

It is essential that the services of a secretary be available to the Cancer Clinic, preferably full time. In addition, a nurse or combination nurse-social worker should be assigned to the Cancer Clinic.

The Cancer Committee should formulate the operational plan for the Cancer Clinic and have it approved by the medical staff and governing board. This plan should define in detail such items as the purpose, organization, personnel, conferences, source and disposition of patients, relationship to other organizations, and other pertinent information concerning the operation of the Cancer Clinic. Once approved by the hospital medical staff, it should be published for the guidance of all concerned.

It is advisable that the above operational plan for the Cancer Clinic, already approved by the hospital staff, then be submitted to the County Medical Society for its approval.

If financial aid is desired in the establishment and operation of the proposed Cancer Clinic, it is necessary that the Clinic plans also be submitted for approval to the Florida Cancer Council after approval by the local County Medical Society. Such financial aid is obtainable for certain phases of clinic operation, etc., from the Florida State Board of Health and from the Florida Division of the American Cancer Society as follows:

Under the present arrangement the Florida State Board of Health may assist in the salaries of personnel or in providing equipment.

The Florida Division of the American Cancer Society may assist in salaries of personnel and may provide funds for supplies and expendable equipment.

Following the American College of Surgeons' Standards, Cancer Clinics may be classified as follows: (1) Cancer Clinics offering full diagnostic and therapeutic facilities, (2) Diagnostic Cancer Clinics, and (3) Hospital departments conducting acceptable cancer programs.

Meeting and Procedure

Each clinic shall have regularly scheduled periodic meetings and shall have a minimum attendance of 80 per cent of the regular members. Consultative members shall maintain 80 per cent attendance when notified of presentation of cases in their specialties.

After diagnosis has been established and recommendations made, those patients without a family physician, and those not eligible for state aid, shall be directed to a panel of approved physicians. Voluntary patients with a family physician, and patients referred to the clinic by private physicians, shall be returned to the physicians concerned with the diagnosis and recommendations of the tumor clinic staff.

Clinics must keep clinical records, cross indices and follow-up files on each case, and these files

should be in accordance with the standards set up by the American College of Surgeons.

All data compiled and recorded by clinics should be made available to authorized representatives of the American College of Surgeons or other supporting agencies if occasion arises.

Cases should be presented at clinic meetings before any therapy has been instituted when feasible.

All cases of tissue diagnosis of tumors presented at approved Cancer Clinics should be submitted with pertinent data to the central tumor registry of the State Board of Health.

The diagnostic nomenclature used by the registry should be in conformity with that recommended by the American College of Surgeons.

General Plan of Operation

Under ideal circumstances, all new tumor cases should come before the staff on first admission to the clinic (either as in-patient or out-patient). However, in large institutions this would tend to overburden the staff with non-problematical cases, and in all institutions might be productive of delay. Hence, a working plan for the average institution might be as follows:

Cases in which surgery is the accepted mode of treatment and which are in prompt need of such (e.g. annular tumor of descending colon) do not need routine presentation in persons. A filled out "approved case record" will be sufficient.

Cases in which radio therapy is the accepted mode of treatment (e.g. small cutaneous angiomata) could be handled similarly. The correctly filled out case record form would be furnished for notification purposes.

Cases in which operation or irridation *might* be but are not unquestionably indicated (either alone or in combination), (e.g. a Stage II carcinoma of the breast) should be shown.

All problem cases in which the procedure is not clear cut should be presented at conference.

The actual therapeutic results, immediate and remote, should be carefully analyzed at regular intervals.

Case records forms should be those as already adopted by the American College of Surgeons or those records showing diagrammatic sketch picturing the lesion and its location. These complete cancer records should be kept available by the tumor clinic secretary.

Follow-up: In order to properly evaluate a cancer program cases should be followed up for a period of at least five years. Many well organized clinics are able to follow up 90 per cent of their cases for this period or longer.

Diagnostic Clinics

The cancer diagnostic clinic is a cancer clinic adapted to the needs of communities and hospitals where the case load is not sufficient to justify full therapeutic equipment or where such equipment is not available. Organization, personnel, conferences, records, and methods of handling patients should be planned as nearly as possible along the same lines as outlined for fully equipped clinics. The cancer clinic staff meets at stated times, acts in a consultative and diagnostic capacity and makes recommendations for treatment. For such a group, close affiliation with some nearby treatment center, to which patients requiring radiation therapy or special surgical procedures will be customarily sent, is desirable. After such treatments these patients can be followed up in the local clinic and an interchange of reports on the therapy used and the notes of the subsequent follow-up will complete the records of both clinics.

In smaller hospitals employing part time pathologists and radiologists arrangements usually can be made to hold the clinic at a time when both men are at the hospital. In a small community the appointment of a permanent staff to the clinic is fully as important as in a large hospital.

YOUR BLUE SHIELD

Blue Shield Annual Meeting

The annual meeting of Active Members of Blue Shield of Florida, Inc., will be held on Sunday, April 27, 1952, at 10:00 a.m. in the northeast dining room at the Hollywood Beach Hotel, Hollywood, in conjunction with the annual convention of the Florida Medical Association. This is an excellent opportunity for Blue Shield physicians to learn first-hand the problems and activities of their plan, and to have a voice in the affairs of the corporation.

Election of Directors

According to the By-Laws of the Plan, the Board of Directors is made up of eighteen directors, chosen as follows: Two participating doctors from each medical district of the Florida Medical Association; two or more participating doctors from the Florida Medical Association's membership at large, and one or more laymen from each medical district

of the Florida Medical Association. All directors are elected by the Active Members of the Corporation and serve for a period of three years each. At the first meeting of the Corporation directors were elected to serve for terms of one, two and three years in order that an equal number of terms would expire each year thereafter.

The following vacancies must be filled this year from the respective medical districts: "A"— one doctor, "B"— one doctor and one layman, "C"— one doctor and one layman, and "D" — one doctor and one layman.

It is not too late for participating physicians who are not now Active Members to make application to the Plan to serve in this capacity, and thereby become eligible to attend the annual meeting. Active membership requires that no dues be paid and does not obligate the physician in any way.

STATE BOARD OF HEALTH

Psittacosis

Psittacosis is primarily an avian infection, but occurs sporadically and in epidemics among the human population. The etiologic agent in birds and man is an obligatory intracellular parasite which belongs to a group of viruses including the infective agents of lymphopathia venereum, meningopneumonitis, and murine and feline pneumonitis. The host range of psittacine virus includes psittacine birds, canaries, pigeons, sea gulls, chickens, ducks, and other fowl.

The avian virus may be transmitted to man in several ways. The commonest mode of transmission is by direct contact with diseased birds or objects contaminated by excreta. Apparently healthy birds may be carriers and transmit the disease. Other modes of transmission include indirect transfer by air and also bite wounds.

The incubation period of psittacosis varies from seven to fourteen days with an average of ten days. The onset may be sudden with chilly sensations, fever, anorexia, sore throat, general malaise, and severe headache, or the presenting symptoms may be vague with a gradual and insidious onset. The disease has most often been confused with influenza, bronchopneumonia and typhoid fever.

Pulmonary involvement is an essential feature of human psittacosis except in especially mild cases. Focal pneumonia usually becomes established within the first few days of illness. Roentgen examination reveals a faint, usually circular, cloud of uniform density situated most often in one of the lower lobes. Scattered, patchy areas of consolidation frequently give this disease the appearance of a migrating pneumonia. Cough and expectoration are usually absent or slight. Nausea and vomiting are common. A relative bradycardia is a characteristic feature. Leukopenia is present in about 25 per cent of cases.

The diagnosis is based on history, symptoms, roentgen study and complement fixation test, and animal inoculation which can be done by the CDC Virus Laboratory through the State Board of Health Laboratory. Physicians desiring that specimens be examined for virus study should mark on the laboratory request slip that the virus examination is desired.

The death rate in human psittacosis has been markedly reduced since the introduction of penicillin and other antibiotics. The beneficial effects of aureomycin, terramycin and penicillin have been demonstrated.

This review of the latest information about psittacosis will be of particular interest to physicians because of the discovery, confirmed by laboratory tests, of psittacosis in fourteen psittacine birds in Dade and Broward counties during the past few months. Also two human cases are reported to have occurred in persons who contracted the disease from a bird purchased in Florida. Of interest to the profession is the fact that the victims were a physician and his wife. The medical profession of the state will undoubtedly want to exercise vigilance in recognizing any possible human cases. It is suspected that the disease among birds has been present in the area for some time without being recognized. It is fortunate that the antibiotic drugs constitute a satisfactory remedy for this serious and often fatal disease.

Study of Indigency of Persons Receiving State Aid for Treatment of Cancer

Frequent inquiries have been made concerning the methods and criteria used by the State Board of Health in determining to whom state aid should be given in its Cancer Control Program. For this reason a study of this subject was made during the fall of 1951. The study was directed by a qualified social worker, Mrs. Lamar Rozear of Jacksonville. For economy reasons it was decided to confine the survey to one hundred consecutive and unselected

cases which were certified in August 1951. These cases fell in thirty-nine different counties from Escambia to Monroe and ranged in population from the smallest to the largest.

Methods of determining indigency vary from county to county. The primary responsibility for determining indigency is placed upon the county health officer but he is encouraged to seek the assistance of state and local welfare agencies and the physicians who are treating the patient. Mrs. Rozear secured the assistance of qualified social workers and made a thorough study of the financial resources of every case that could be found. Such factors as current income, real estate, life insurance, automobiles, etc., were looked into.

The following is a summary of the findings made:

Incomes of Patients Fully Studied

Single persons Income $75 or less	4
Families (2 or more) $150 month or less	33
Families (2 or more) $150-200 monthly	5
Families (2 or more) $200-250 monthly	4
Families (2 or more) $250-325 monthly	5
Unknown	2
Public assistance recipients	40
Unable to locate	4
Deceased (financial circumstances unknown)	3
Total	100

The conclusion reached was that there were few, or no, persons in this group where medical indigency could be considered in doubt. The five patients with family incomes ranging from $250 to $325 monthly all were members of large families. Since forty of the one hundred persons had already been thoroughly investigated by the State Welfare Board it was not felt worthwhile to repeat their work.

Although the methods employed by the State Board of Health and county health departments for determining indigency are crude, and investigations are not made routinely by trained social workers, it is felt that it would not be worthwhile from a financial standpoint to change the methods now being used. It is estimated that it would cost at least $50,000 per year to have all prospective state aid patients investigated by social workers whereas the entire budget for Cancer Control is only about four times that amount. It is believed, however, that an occasional check such as that described above by qualified social workers is indicated. The survey that was made cost only about $1,200. A similar study is now being made of persons receiving insulin for diabetes .

BIRTHS AND DEATHS

Births

Dr. and Mrs. Leonard Garten of Jacksonville announce the birth of a son, Barry Arthur, on December 21.

Dr. and Mrs. George F. Schmitt, Jr., of Miami announce the birth of a daughter, Susan Lynn, on January 12.

Deaths — Members

Snow, Henry O., Tampa ..... Feb. 18, 1952
McDuffie, Harold F., Jacksonville ..... March 4, 1952

Deaths — Other Doctors

Buckner, Rufus G., Tampa ..... Recently
Evans, Kline W., Columbia, Tenn. ..... Recently
Hilborn, Robert R., Midland, N. C. ..... Nov. 16, 1951

NEW MEMBERS

The following doctors have joined the State Association through their respective county medical societies.

Alsup, Fred W., (Col.), St. Petersburg
Anderson, James R., West Palm Beach
Bane, Russell C., St. Petersburg
Boyce, J. Clifford, Sanford
Bray, Walter A., St. Petersburg
Brown, Clyde M., Tampa
Buford, Edward T., Jr., (Col.), Panama City
Burns, Edward C., Jr., Lake Wales
Croom, William C., Jr., Jacksonville
Curry, Hiram B., Jasper
Debo, Raymond A., Eustis
Frazier, Truett H., Orlando
King, Samuel A., Avon Park
Knauer, William J., Jr., Jacksonville
McKell, Thomas E., Tampa
Meadows, Benjamin J., Jr., Dade City
Pope, Charles L., Tampa
Rogers, Lee, Jr., Cocoa
St. John, Joseph H., Jacksonville
Schoetker, George H., Clearwater
Smith, Lynwood B., Tampa
Tomlinson, Wray J., Jacksonville
Vaughan, Davis H., Clearwater
Wadsworth, Charles L., Ft. Lauderdale
Winstead, George A., Panama City

STATE NEWS ITEMS

The Florida Medical Association Smoker Committee for the annual meeting in April met in Hollywood, Sunday, February 24, with 100 per cent attendance. Present were Drs. Reuben B. Chrisman, Jr., chairman, Miami; Francis T. Holland, Tallahassee; Chas. J. Collins, Orlando; Lewis T. Corum, Tampa; and Curtis H. Sory, Fort Lauderdale. Also in attendance was Dr. Stewart G. Thompson of Jacksonville, managing director.

Dr. Joshua C. Dickinson of Tampa was elected Chancellor of the American College of Radiology at the annual meeting on February 8 in Chicago.

Dr. Walter C. Jones of Miami, president-elect of the Southern Medical Association, attended the meeting of the executive committee of the Council of the Southern Medical Association at the Hotel Nacional, Havana, Cuba, January 24. A diploma which expresses the esteem in which the members of the Southern Medical Association are held by the physicians of Cuba and an appreciation of the professional brotherhood between physicians of the United States and Cuba was presented to each officer of the Southern Medical Association attending the meeting, by the Cuban Medical Association and the Havana Medical Association.

Dr. John P. Michaels of Orlando was the main speaker at a recent program meeting of the Pilot Club of that city. Dr. Michaels, president of the Central Florida Mental Hygiene Society, told the history of the society and stressed the need for education to aid in the mental problem.

Dr. Herbert L. Bryans of Pensacola was re-elected president of the Florida State Board of Health for a twelfth term at the board's annual session in Jacksonville, February 11-13.

Dr. Jack Q. Cleveland of Coral Gables represented Florida in Chicago, February 17, at the "Kick-off" Conference of the 1952 drive for funds by the American Medical Education Foundation.

Dr. Nathan Weil, Jr., of Jacksonville attended a course on the care of the Premature Infant at New York Hospital in January.

Two members of the Florida Medical Association have been appointed by State Superintendent of Public Instruction, Thomas D. Bailey, to a newly formed state advisory committee on practical nursing to assist in operation of a practical nurses training program in the state. Representing the Florida State Board of Health is Dr. Herbert L. Bryans of Pensacola, and representing the Florida Medical Association is Dr. Jere W. Annis of Lakeland.

Dr. George R. Gage of Miami spoke to members of the Cerebral Palsy Parents Council of the Dade County Society for Crippled Children recently at the clinic.

Dr. Joseph L. Kinzie of Lake Wales was among speakers who discussed their professions at the Career Day program at Lake Wales High School recently. In discussing the profession of medicine, Dr. Kinzie stressed the fact that a doctor's time is never his own, and that he must be ready to give his time, have an interest in people, and want to help them.

Dr. Joseph S. Stewart of Miami will preside at the joint meeting of the Southeastern Surgical Congress and The Atlanta Graduate Medical Assembly in Atlanta, March 10-13. Registration for both assemblies is $15. For information, write Mrs. S. R. Roberts, Executive Secretary, 768 Juniper St., N.E., Atlanta.

The Second Annual Seminar of the Dade County Pediatric Society will be held at the Jackson Memorial Hospital, Miami, April 23-25, 1952. Registration fee for non-members is $10. For information write Dr. W. S. Nock, 273 Alhambra Circle, Coral Gables.

Dr. James C. Lanier of Jacksonville spoke on various phases of pediatrics at a recent meeting of the Southside Junior Woman's Club of that city.

Dr. Robert O. Burry of DeLand answered questions about diseases of the heart on a recent special program over radio station WJBS. The address was one of a series arranged in conjunction with the Heart Drive.

Dr. Roger E. Phillips of Orlando discussed a medical film, "That Angry Boy," at the regular monthly meeting of the Florida Nurses Association, District 48, in January.

Dr. Ernest R. Bourkard of Tampa spoke on "Electric Shock Therapy," at the regular January meeting of the Tampa Bay Psychological Association.

The Eighteenth Annual Meeting of the American College of Chest Physicians will be held at the Congress Hotel, Chicago, June 5 through 8, 1952. A scientific program covering all recent developments in the treatment of heart and lung disease is being arranged.

Dr. M. Jay Flipse of Miami is Regent of the College for the district and Dr. Clarence M. Sharp of Jacksonville serves as Governor of the College for Florida. Officers of the Florida Chapter of the College are Dr. Howard K. Edwards, president, and Dr. Nathaniel M. Levin, secretary-treasurer, both of Miami.

The Board of Examiners of the American College of Chest Physicians announces that the next oral and written examinations for Fellowship will be held in Chicago on June 5, 1952. Candidates for Fellowship in the College who wish to take the examinations should contact the Executive Secretary, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11.

Dr. Carlos P. Lamar of Miami attended the International Conference on Vitamins and Metabolism held in Havana, Cuba, February 23-26. The Conference was sponsored by the University of Havana in conjunction with Northwestern University, Chicago, and the Nutritional Clinic, Birmingham, Ala.

Dr. James T. Hardy of Lake Worth spoke on the various types of heart conditions and their symptoms at a recent meeting of the Carl Vogel-Donald Lee Post 47, American Legion, of Lake Worth.

Dr. Joseph L. Selden, Jr., of Fort Myers spoke at a recent meeting of the Lee County Cancer Society.

Dr. Joe F. Garner, formerly of Jacksonville, announces the opening of his offices in Dothan, Ala.

The Committee on Medical Motion Pictures has completed the 1951 supplement to the second revised edition of the booklet entitled "Reviews of Medical Motion Pictures." This supplement contains 90 reviews of medical and health films reviewed in The Journal of the A. M. A. from Jan. 1, 1951, through Dec. 31, 1951. Each film has been indexed according to subject matter. The purpose of these reviews is to provide a brief description and an evaluation of motion pictures which are available to the medical profession.

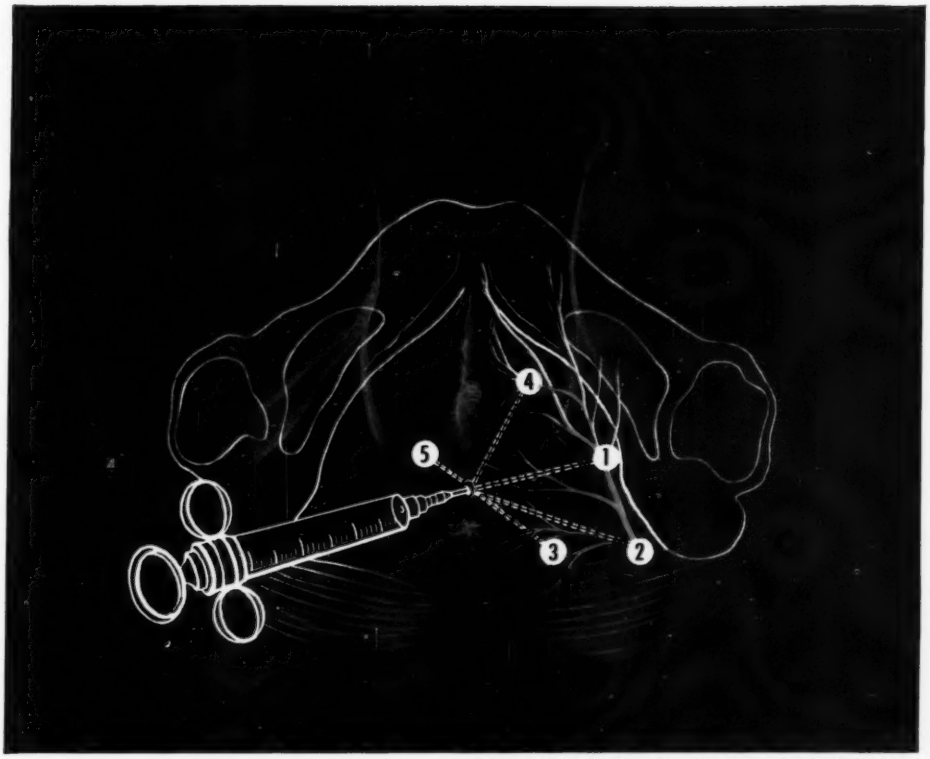
A study of children's diseases and their occurrence in Florida has been initiated by the Florida Heart Association and the Florida Pediatric Society. A committee to undertake the study has been appointed by Dr. Egbert V. Anderson of Pensacola, president of the Florida Pediatric Society, and Dr. Elwyn Evans of Orlando, president of the Florida Heart Association. Members of the committee are Drs. Edward L. Cole, Jr., and James L. Gouaux of St. Petersburg; Lewis T. Corum and H. Phillip Hampton of Tampa; and James M. Stem of Clearwater and John A. Mease, Jr., of Dunedin.

Dr. Edward R. Annis of Miami spoke at the second annual banquet of the ten classified women's service clubs of the Miami area. His subject was "Brains in the Stockpile."

Dr. Benjamin F. Barnes of Chattahoochee was presented a plaque by the Woodmen of the World at an oyster supper given by the Chattahoochee Magnolia Camp 228. The plaque award was presented to Dr. Barnes in recognition of his outstanding service as a doctor and citizen in Chattahoochee for the past 50 years.

Dr. William M. Davis of St. Petersburg, who has practiced in that city longer than any other physician in Pinellas County, recently marked the forty-fifth anniversary of his start in medicine in St. Petersburg.

(*Continued on page 740*)



Sites for injection of local anesthesia in obstetrics. Sites 1 to 4 should be similarly injected on the contralateral side. Site 5 is for episiotomy. Adapted from Johnson, O. J.: Nerve Block in Painless Childbirth, J.A.M.A. *145*:401 (Feb. 10) 1951.

Pudendal Block in Obstetrics Simplified with **ALIDASE**

Using a local anesthetic with hyaluronidase, Heins[1] reports: "Complete perineal anesthesia is practically instantaneous. . . . The technique of pudendal block is greatly simplified. The operator does not have to inject the nerve per se, but infiltration in the vicinity of the nerve will accomplish an effective block."

Baum[2] states: "The use of hyaluronidase is found to be a safe and simple method for increasing the efficiency of pudendal block in obstetrics and for overcoming many of the objections to this type of obstetrical anesthetic."

ALIDASE®—highly purified, well tolerated brand of hyaluronidase—definitely shortens the period between completion of the block and establishment of operating analgesia. Swelling, induration and discomfort are almost negligible with Alidase.

[1]Heins, H. C.: Pudendal Block with Hyaluronidase, J. South Carolina M. A. *46*:309 (Oct.) 1950.
[2]Baum, F. E.: The Use of Hyaluronidase in Pudendal Block, Am. J. Obst. & Gynec. *60*:1356 (Dec.) 1950.

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

RESEARCH IN THE SERVICE OF MEDICINE **SEARLE**

Therapy for Vascular Headache to Reverse the Physiologic Disturbance

Headache, a problem encountered in all kinds of medical practice, may occur in association with any of a variety of disorders, some organic, other purely functional.

Among the several types, functional headaches present the greatest problem because of their obscure etiology and recurrent nature.

Among these are:

Migraine (both classical and variant forms)
Tension headache
Psychogenic headache
Histaminic cephalgia

Wolff and his co-workers established that the pain of these headches is due to disturbance of the tonus of cranial blood vessels — hence the term *vascular headaches.*

The craniovascular changes associated with the several phases of the typical migraine attack are:

Vasoconstriction — to which the visual prodromata are attributable. It is possible to abort the attack during this phase in all but a few cases. (See treatment below.)

Vasodilatation — as the vessels lose their tone, exaggerated pulsations set in, resulting in the throbbing pain which characterizes vascular headache. Treatment for the attack is still effective during this phase. (See below.)

Vessel Edema — if the vasodilation continues for too long, vessel walls become edematous; this changes the character of the pain to a steady, intense aching. The attack can now no longer be checked, even with maximum dosage of specific drugs. *Moreover, sustained headache often induces reflex neck muscle tension, a source of residual pain.*

Therapy: 1. Reduce the frequency of attacks — psychotherapy and regulation of living habits to avoid fatigue and nervous tension.

2. Relieve the acute attack — of the numerous drugs which have been tried, ergotamine and its derivative preparations have proved most effective. The newest product is oral tablets of Cafergot®, N. N. R. (ergotamine with caffeine 'Sandoz'). When dosage is adjusted to the needs of the individual, Cafergot will give good relief in 85% of cases. It enables a greater number of patients to benefit from early administration since the oral route simplifies treatment as compared to parenteral therapy.

The dosage procedure is:

1. Take 2 tablets at first sign of the attack.
2. If attack continues, take one additional tablet every ½ hour until attack is terminated *(max. 6 tabs. per attack).*

Many migraine patients delay taking medication until the attack is at its height. Explicit dosage instructions may be forgotten unless the patient comes to realize their importance. Therefore, to encourage adherence to correct procedure, we have prepared pads outlining detailed dosage instructions. Supplies of these INSTRUCTION SLIPS will gladly be sent upon request.

GENERAL REFERENCES: DeJong, R.: Chicago M. Soc. Bull. *54:* 106, 1951. Friedman, A.: Modern Headache Therapy, St. Louis, C. V. Mosby Co., 1951. Wolff, H.: Headache and Other Head Pain, N. Y., Oxford Univ. Press, 1948.

Sandoz Pharmaceuticals
DIVISION OF SANDOZ CHEMICAL WORKS, INC.
68 CHARLTON STREET, NEW YORK 14, N. Y.

(Continued from page 738)

Dr. Paul F. Hutchins of Jacksonville is at Tulane University of Louisiana School of Medicine for a year's study in Orthopedic Surgery and Adult Reconstruction.

Dr. Oscar L. Kelley of Palm Beach spoke on "Practical Aspects of Geriatric Nursing" at a recent semi-monthly meeting of Licensed Practical Nurses, Division 1.

WANTED — FOR SALE

Advertising rates for this column are $5.00 per insertion for ads of 25 words or less. Add 20c for each additional word.

LOCATION DESIRED: Surgeon, F. A. C. S. and Board eligible, age 37, desires Florida location. Will consider association with older surgeon, community needing general surgeon or industrial position permitting some private practice. Capable and well trained. Best of references. Florida license. Write 69-62, P. O. Box 1018, Jacksonville, Fla.

FOR SALE: Florida — Jacksonville — Lucrative E. E. N. T. practice for sale by Estate. Established over forty years. Equipped offices in downtown building. Write 69-63, P. O. Box 1018, Jacksonville, Fla.

PHYSICIAN WANTED: White, gentile, American born, protestant, grade A graduate, internship and at least one year surgical residency. Real opportunity with rapid advancement. Located in Central Florida. Write 69-64, P. O. Box 1018, Jacksonville, Fla.

PHYSICIAN WANTED: Resident House Physician required at New England's largest summer resort hotel, the Mount Washington Hotel, Bretton Woods. New Hampshire. Season—July 1st to September 10th. Please communicate with Oscar T. Johnson, Managing Director, at Homosassa Springs Hotel, Homosassa Springs, Fla.

PHYSICIAN WANTED: Young or middle aged, general practitioner, for county-seat town. Reason for needing physician: My age and heavy work. Write O. F. Green, M.D., Mayo, Fla.

NOTICE

Your March Journal carried a complete program and other detailed information relative to the Seventy-Eighth Annual Meeting of the Association in Hollywood, April 27-30.

COMPONENT SOCIETY NOTES

Bay

At the regular meeting of the Bay County Medical Society in February, Dr. George S. Tootle, surgeon from Hartford, Ala., reported on the meeting of the American College of Surgeons held in Brimingham in January and presented some of the information and ideas developed there.

Dade

At the March meeting of the Dade County Medical Association, papers were presented on "Pitfalls of 'Routine' X-Ray Examination." Dr. Richard D. Shapiro spoke on "The Chest;" Dr. Oliver P. Winslow, "The Skeleton;" Dr. Maurice M. Greenfield, "The Abdomen;" and Dr. Eugene H. Bryson, "The Gastro-Intestinal Tract."

Duval

At the regular monthly meeting of the Duval County Medical Society in March, Drs. Eugene M. Frame, J. K. David, Jr., and Ivan Isaacs presented a paper on "Discussion of Scurvy as Seen in Jacksonville Area."

Escambia

Members of the Escambia County Medical Society were among the speakers at a five-day institute on nursing care for the polio patient held March 3-7 at Crippled Children's Home in Pensacola.

Lake

At the regular monthly meeting of the Lake County Medical Society in February, Drs. Lorenzo L. Parks and William A. Walter of the Florida State Board of Health, Jacksonville, spoke on "Serology Tests." The wives were guests of the members at this meeting, and the organization of the Woman's Auxiliary was completed.

Monroe

Members of the Monroe County Medical Society met with the medical staff of the U. S. Naval Hospital at Key West for the regular February meeting. Dr. D. J. Leithauser of Detroit spoke on "Early Ambulation in the Prevention of Postoperative Pulmonary Embolism," and the paper was discussed by Dr. John M. T. Finney, associate professor of surgery, Johns Hopkins University School of Medicine.

(*Continued on page 742*)

Rx

...FOR BOTH DOCTOR AND PATIENT

• **The name Sealtest is your guarantee of rich, wholesome dairy foods—healthful refreshment at its nourishing best.**

Southern Dairies
Sealtest
ICE CREAM

(*Continued from page 741*)

Pinellas

Mr. Jefferson Hamilton addressed the Pinellas County Medical Society at its regular monthly meeting on March 3. His subject was "The Proposed University of Florida Medical Center."

Dr. Wesley W. Wilson of Tampa spoke on Dermatology at the April meeting.

Members of the Pinellas County Medical Society recently were invited to a special showing of the professional film, "Uterine Cancer: The Problem of Early Diagnosis," at the Pinellas County Tumor Clinic. The film was brought to St. Petersburg by the American Cancer Society.

Sarasota

At the regular February meeting of the Sarastoa County Medical Society, the Society endorsed the campaign of the Sarasota Heart Committee, which was conducting a fund-raising drive in cooperation with the American Heart Association.

Seminole

All members of the Seminole County Medical Society have paid their state dues for 1952.

Volusia

Dr. John J. Cheleden of Ocala addressed the regular meeting of the Volusia County Medical Society in Daytona Beach in February.

SEND THIS COUPON TODAY

for AVEENO . . . the new concentrate from oatmeal . . . for skin therapy.

Aveeno provides prolonged emollient action on irritated skin areas . . . controls pruritus ani and pruritus vulvae . . . relieves itching and skin irritation in general rashes, sulfa and penicillin rashes, eczemas and allergic reactions.

E. FOUGERA & CO., INC.
75 VARICK ST., NEW YORK 13, N. Y.
Please send professional samples of **AVEENO®.**

_____ M.D.
ADDRESS _____
CITY _____ STATE _____

HILL CREST SANITARIUM
FOR NERVOUS AND MENTAL DISEASES AND ADDICTIONS

Insulin and Electro-Shock Therapy Used in Selected Cases. Gradual Reduction Method Used in the Treatment of the Addictions.

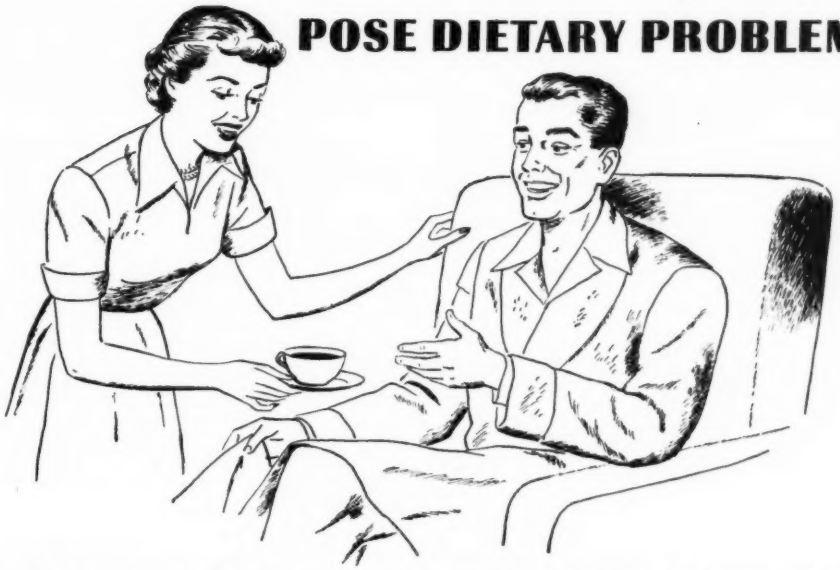
Thoroughly modern in architecture and construction. Eight departments — affording proper classification of patients. All outside rooms attractively furnished. Several bathrooms and rooms with private bath on each floor. Also a spacious sun parlor in each department. Located on the crest of Higdon Hill, 1,050 feet above sea level, overlooking the city, and surrounded by an expanse of beautiful woodland. Ample provision made for diversion and helpful occupation. Adequate night and day nursing service maintained. Catalogue sent on request.

James A. Becton, M. D., Physician-in-charge
P. O. Box 2896, Woodlawn Station, Birmingham, Alabama

James Keene Ward, M. D., Associate Physician
Phones 9-1151 and 9-1152

When Functional Derangements

POSE DIETARY PROBLEMS



In the interest of maintaining good nutrition in the patient, many functional derangements of the gastrointestinal tract make the use of a well rounded dietary supplement, such as Ovaltine in milk, highly advantageous. Among such functional derangements more commonly encountered are nausea, anorexia, gastritis, diarrhea, dysentery, enteritis, and colitis.

In these conditions, Ovaltine in milk is particularly useful, not only because of its easy digestibility but also because of its blandness and its high nutrient content. It offers the opportunity of providing a balanced fare of essential nutrients without mechanical irritation or excessive digestive demands. Hence it qualifies especially when customarily eaten foods are contraindicated and a nutritious bland diet is required.

The wealth of nutrients supplied by three glassfuls of Ovaltine in milk is outlined in the table below.

THE WANDER COMPANY, 360 N. MICHIGAN AVE., CHICAGO 1, ILL.

Ovaltine

Three servings of Ovaltine, each made of ½ oz. of Ovaltine and 8 fl. oz. of whole milk, provide:

PROTEIN	32 Gm.	VITAMIN A	3200 I.U.
CARBOHYDRATE	65 Gm.	VITAMIN D	420 I.U.
FAT	30 Gm.	ASCORBIC ACID	30 mg.
CALCIUM	1.12 Gm.	NIACIN	6.7 mg.
COPPER	0.7 mg.	PANTOTHENIC ACID	3.1 mg.
IODINE	0.7 mg.	PYRIDOXINE	0.6 mg.
IRON	12 mg.	RIBOFLAVIN	2.0 mg.
PHOSPHORUS	940 mg.	THIAMINE	1.2 mg.
CALORIES	658		

Two kinds, Plain and Chocolate Flavored. Serving for serving, they are virtually identical in nutritional content.

Allen's Invalid Home
MILLEDGEVILLE, GA.
Established 1890
For the treatment of
NERVOUS AND MENTAL DISEASES
Grounds 600 Acres
Buildings Brick Fireproof
Comfortable Convenient
Site High and Healthful
E. W. ALLEN, M.D., *Department for Men*
H. D. ALLEN, M.D., *Department for Women*
Terms Reasonable

Ambulance Service
FERGUSON FUNERAL HOME
H. E. Ferguson, Licensed Director
1201 South Olive
WEST PALM BEACH, FLA.

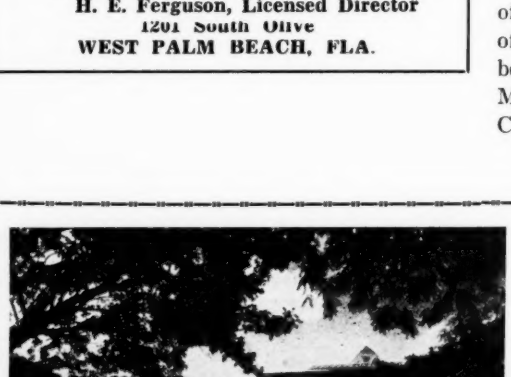
OBITUARIES

John A. Stanford

Dr. John A. Stanford of Fort Lauderdale died at his home there on Dec. 27, 1951, after a lingering illness. He was 76 years of age.

A native of Florida, Dr. Stanford was born in Columbia County on April 26, 1875. He received the degree of Doctor of Medicine from the Medical College of the State of South Carolina at Charleston in 1909. Immediately thereafter, he returned to Florida and began the practice of medicine in Dunnellon. Four years later he moved to Fort Lauderdale, becoming one of the first physicians to serve that area. At that time, Fort Lauderdale was little more than a flag station on the railroad, headquarters for the Bass Fisheries and a trading post for the Seminole Indians. Soon the Florida East Coast Railway appointed Dr. Stanford as railroad physician, a post which he held for many years. As a general practitioner, he pursued his profession faithfully and well and was held in high esteem by early settlers and Indians alike. After twenty-nine years of medical practice in his native state, he was forced to retire in 1938 because of ill health.

Being civic-minded, Dr. Stanford was active in promoting the growth of Fort Lauderdale. As early as 1925 he served as president of the First National Bank and for many years was a member of its board of directors. He also was vice president of the Pioneer Department Store. A charter member of the local Elks Lodge, he was in addition a Mason and a member of the First Methodist Church, where for years he sang in the choir.



MIAMI MEDICAL CENTER
P. L. DODGE, M. D.
Medical Director and President
1861 N. W. South River Drive
Phones 2-0243 — 9-1448
A private institution for the treatment of nervous and mental disorders and the problems of drug addiction and alcoholic habituation. Modern diagnostic and treatment procedures — Psychotherapy, Insulin, Electroshock, Hydrotherapy, Diathermy and Physiotherapy when indicated. Adequate facilities for recreation and out-door activities.
Information on request
Member American Hospital Association

When the Broward County Medical Society was organized in April 1926, Dr. Stanford was a charter member. Two years later he served as its president. Following his retirement from active practice, he became an honorary member of this society and also of the Florida Medical Association and the American Medical Association.

Survivors include two daughters, Miss Vera Stanford and Mrs. Alvarez Putt of Fort Lauderdale; two sons, Dr. H. B. Stanford of Hartwell, Ga., and Kramer Stanford of Miami; and three grandsons, John W. Stanford, a student at the University of Florida, and James Sterling Whisenhunt and Neil Stanford Whisenhunt, both of Fort Lauderdale.

James Fling Pitman

Dr. James Fling Pitman died on Jan. 31, 1952 at his home in Lake City after a long illness. He was 61 years of age.

Born in LaGrange, Ga., on June 14, 1890, Dr. Pitman was educated in his native state. In 1913 he received his medical degree from the Atlanta College of Physicians and Surgeons. During World War I he served as a captain in the United States Army.

After practicing for a number of years in Atlanta, Ga., Dr. Pitman moved to Lake City in 1933 to become associated with the Veterans Administration Hospital there. Radiology was his specialty, and in 1942 he resigned from the Veterans Administration to enter the private practice of radiology in Lake City.

In war and in peace, Dr. Pitman served his community, his state and his nation well and was held in highest esteem by his fellow citizens and his colleagues. He was a member of the Veterans of Foreign Wars, the American Legion and the Lake City Rotary Club.

Dr. Pitman was a past president of the Columbia County Medical Society, in which he had held membership for fifteen years. He was a member of the Florida Medical Association and the American Medical Association. A past president of the Florida Radiological Society, he was also a fellow of the American College of Radiology and the Radiological Society of North America.

Surviving are the widow, Mrs. Fannie Oliver Pitman, and a son, James H. Pitman, both of Lake City; a daughter, Mrs. C. C. McMurtry of Kansas City, Mo.; and a sister, Miss Anna May Pitman of Bangor, Me. Two grandchildren also survive.

Advertisement

From where I sit *by* Joe Marsh

They Do "Give A Hoot" For Easy

Easy Roberts finally got rid of the noisy pigeons that used to whoop it up under his eaves.

He must have tried a dozen ways to scare them off. But no matter what he did, they would be right back cooing by his window the next morning.

Then Easy thought of an old stuffed owl he had in his attic. He propped it on the roof so's all the pigeons could see it. They left . . . and three hoot owls have taken their place. Easy swears the hooting is even worse than the cooing of the pigeons.

From where I sit, bright ideas often turn out to be "not so bright." That's why we shouldn't be too positive about our own opinions. Some people like to tell their neighbors who to vote for, how to practice their profession, even what beverage to choose. I believe a glass of beer is the best thirst-quencher—you may believe differently. But who's to say one's right and the other is wrong? Let's just practice tolerance. It'll save a lot of hootin' and hollerin'.

Joe Marsh

Copyright, 1952, United States Brewers Foundation

The Right to *Choose* . . . .

CORNERSTONE OF AMERICAN LIBERTY

Hospital Expense Plans

Medical-Surgical Expense Plans

Paycheck Protection Plans

Life Insurance Plan

AS long as Americans keep the right to choose — their church, their political party, their doctor and their hospital — freedom must prevail

We are proud that hundreds of thousands of Americans have freely chosen the protection afforded them by PENNSYLVANIA LIFE'S liberal, low-cost health insurance plan.

Pennsylvania Life
HEALTH & ACCIDENT **Insurance Company**

FOUNDED 1890

HOME OFFICE: PHILADELPHIA 5, PA.

19 District Offices in Florida

Miami	Executive Office 3028 Biscayne Blvd.	Fort Lauderdale	521½ South Andrews Avenue
	Downtown 1210 Pacific Building	Jacksonville	303 Clark Building
	Southwest 1260 S. W. 22nd Street	Tampa	228 Cass Street Arcade Building
	Little River 260 N. E. 79th Street, Rm. 3	Sarasota	237 Main Street, Room 15
	Coral Gables 3028 Biscayne Blvd.	Fort Pierce	Florida Bank Building, Room 34
	Miami Beach 3028 Biscayne Blvd.	Daytona Beach	116½ Orange Avenue
St. Petersburg	515 White Building	Tallahassee	429 Centennial Building
Lakeland	206 Marble Arcade	Panama City	142 Harrison Avenue
West Palm Beach	410 Datura Street	Pensacola	501 Theisen Building
Orlando	209 Slayton Building		